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TRANSACTIONS & STUDIES
of the
College of Physicians of Philadelphia

Volume 34
(Fourth Series)

Number 1
(July 1966)

Control of Conception in Primates[1]

By LUIGI MASTROIANNI, JR., M.D.[2]

THE broad title of my lecture allows exploration of the importance of reproductive processes in both *homo sapiens* and the subhuman primate. For the human species we now have demographic data which have brought into focus the dire consequences of unlimited population expansion, both at home and abroad. For the clinician, the importance of conception control is perhaps more meaningfully expressed in terms of the lives of those individuals who reproduce involuntarily or who are involuntarily reproduced. Recently developed methods of family planning, the "pill" and the intrauterine device, have opened the way for large scale programs. It is evident, however, that we do not have at our disposal *the* ideal method of birth limitation. Indeed, we may never have one single method which would be suitable for use in all circumstances. At present the range of choices is narrow, and this, in my opinion, is a reflection of the glaring lack of basic information in primate reproductive biology.

Examples of deficiencies in our knowledge of reproductive processes are numerous. In most primates, including man, one ovum is released monthly from a ripe Graffian follicle. In a given cycle several other follicles become nearly mature and then regress. The basic processes associated with follicular maturesence, ovulation, and follicular regression are not as yet completely understood. Once released, the egg is transferred to the fallopian tube, which serves as a bridge between the ovary and the uterus. There it is fertilized, if this be a conceptive cycle, by one of many spermatozoa, which were deposited in the vagina some hours earlier. The spermatozoa were probably conditioned in the female reproductive tract and have acquired the capacity to penetrate the egg (1). The effect of the environment of the reproductive tract on the spermatozoa and the conditioning influence which precedes fertilization are matters which are not yet fully explored in the primate, nor are these processes fully understood in the rodent. After ovum penetration by one spermatozoan, somehow there is a barrier to further ovum penetration. The process of fertilization, beginning with penetration of the egg by the spermatozoan and ending with the joining of the male and female components (pronuclei) within the egg itself is

[1] James M. Anders Lecture XLIV, The College of Physicians of Philadelphia, 2 March 1966.

[2] Professor and Chairman, Department of Obstetrics and Gynecology, University of Pennslyvania School of Medicine, Philadelphia, Pennsylvania 19104.

TABLE I

PENNSYLVANIA SERIES*

	Non-coil (Controls)	Coil
Number of cycles	10	11
Ovulation points	29	49
Tubal ova	9	0
Uterine ova	0	7

Ovum recovery experiments in superovulated monkeys (macaca mulatta). Fallopian tubes and uterus were flushed within 4–6 hours of the estimated time of ovulation.

not completely understood in the sea urchin, let alone in man.

Once fertilized, the egg develops in the tube where it is contained for about three days. We know that if the egg is released prematurely from the tube it will not survive, but we do not know as yet in man, or in the subhuman primate, how the egg is retained temporarily within the tube. After the egg has been transferred to the uterus it remains free in uterine fluid for about three days, and finally it is embedded in the endometrium. The quality of the secretions of the tube and the uterus in which the developing ovum spends the first six days of life has not been completely explored in man, or for that matter in the rabbit. The processes which bring about attachment of the developing ovum (implantation) are also not clear.

Of the two newer methods of family planning, one, the "pill," was developed as a result of application of information derived from laboratory animal experiments. The story behind the development of this meaningful contribution to population control by Rock, Pincus, Garcia, and others is now well known (2). The other recently developed method, the intrauterine device, arose out of empiric observations carried out initially in patients. We know that the intrauterine device is effective (3), but thus far we have turned to the experimental animal—in this case the subhuman primate—to explain its mechanism of action.

Ovum recovery experiments in the superovulated, artificially inseminated Macaque monkey support the thesis that the presence of an intrauterine device is associated with rapid discharge of eggs from the fallopian tube (4). All of the ova thus far recovered in monkeys with an intrauterine device have been found in the uterus shortly after ovulation, when they should have been still in the fallopian tube. A total of 11 unfertilized ova have now been recovered from the uterus of superovulated, artificially inseminated coil monkeys. Seven of the 11 were recovered in recent experiments at the University of Pennsylvania (Table I). In the control, non-coil animals, no uterine ova were found, and the recovery rate from the tubes was 31%, as opposed to 0% in the coil animals. Experiments are being continued in the monkey to explore the way in which the rapid transfer of ova is brought about. A better understanding of some of the basic processes of ovum transport may very well open the way to new and imaginative methods of family planning. Conceivably, other means can be devised to bring about the premature release of ova from the tube. A pharmacological method to encourage ovum transfer might signal the way for an effective postcoital "pill."

The current lack of basic information in reproductive physiology is now receiving increased attention, thanks to the resources of the recently established N.I.H. Institute for Child Health and Human Development, the Ford and Rockefeller Foundations and other agencies. Training programs have been funded which are now beginning to attract talent to this area. In all of medical research there is, however, a lag period between laboratory discovery

* The efforts of S. Suzuhi, M.D., Y. Manabe, M.D., and F. Watson, W. D., and the technical assistance of S. Shattuck and C. Meltzer are gratefully acknowledged.

and practical application of methods to the patient. This is painfully evident in the field of reproduction—a field in which no delay should be tolerated. It arises, at least in part, out of lack of availability of support for patient oriented research. In addition to family planning programs directed at *patient service,* we need programs directed at *patient oriented research,* possibly with the subhuman primate as the intermediary. Such programs should be located in medical centers which have already in them a hard core of basic scientists. In this way a dialogue could be established between the biochemist and biologist on the one hand and the research oriented clinician on the other. A comprehensive approach to the problem is long overdue, and the delay has been costly.

REFERENCES

1. Chang, M. C.: Fertilizing capacity of spermatozoa, in *Endocrinology of Reproduction.* Ed. by C. Lloyd, Academic Press, New York, 1959.
2. Maisel, A. G.: *The Hormone Quest.* Random House, New York, 1965.
3. Proceedings of the Second World Conference on the Intrauterine Device.
4. Mastroianni, L., and C. Rosseau: Influence of the intrauterine coil on ovum transport and sperm distribution in the monkey. Am. J. Obs. & Gyn. **93**: 416, 1965.

An American Clinician in Asia[1,2]

By THEODORE E. WOODWARD, M.D.[3]

LAHORE is the cultural center of old India located in the Western Punjab and is the present sentimental capital of Pakistan. It is a beautiful city of great contrasts marking the junction of the Grand Trunk Road to Calcutta from the northwestern frontier of the Asian subcontinent and the Ferozepore Road south to Delhi. Kim's cannon, Zam-zammah, which spat thunder throughout the Punjab, now guards the lower Mall across from the Museum.

In this setting, Akbar, Jehangir, and Shah Jahan resided in cooler weather, consolidated their vast Mogul empire and constructed beautiful monuments which still give evidence of their architectural skill. A massive Red Fort, a city within a city, and such edifices as the Badshai Mosque, the Shalimar Gardens and Jehangir's Tomb, are striking examples of beauty and symmetry. Alexander of Macedon extended the frontier of the Greek empire to just east of the Indus where he spent a few of his latter years.

Pakistan (Land of the Free) was born with partition from India in 1947. Comprising approximately one hundred million people of Muslim faith, it is really two countries, the East and West Wings, separated by 1,200 miles of Northern India, south of the Himalayan chain. East Pakistan is a delta, a land of rivers, with certain diseases peculiar to its environment, such as Asiatic cholera and malaria. West Pakistan is arid and semi-arid, depending upon water from its few rivers, a system of canals, and newly constructed dams. The word Punjab means "five rivers," with the head waters of three arising in India-held Kashmir. These rivers form the Indus, site of one of our early civilizations.

In this rapidly developing country, health, a burgeoning population, malnutrition, and water are immediate major problems. A changing influence on the health practices of the masses will take time, since educational opportunities will only slowly reach the poor. Newly organized rural health centers and better agricultural practices must painstakenly elevate the health and living standards of villagers. The problem of salinity, or water trapping by salt-laden alluvial soil, must be solved to provide more sorely needed arable land. The United States has made significant contributions to this new country in certain of these areas. There are no less than five American universities involved in Pakistan at present: University of Washington, University of Chicago, University of Southern California, University of Indiana, and University of Maryland.

Infectious diseases are the prime cause of death in Pakistan. Population density and protein malnutrition create an environment for the spread of disease and substandard health conditions in poverty-stricken areas in Lahore and elsewhere.

[1] Benjamin Musser Lecture IX, The College of Physicians of Philadelphia, 6 April 1966.

[2] From the Department of Medicine and the Institute of International Medicine, University of Maryland School of Medicine, the Children's Hospital of the Fatima Jinnah Medical College, Lahore, and the East Medical Service, Mayo Hospital, Lahore, Pakistan.

This study was supported in part by Research Grant No. 11327 entitled, International Center for Medical Research and Training from the Office of International Research, National Institutes of Health of the United States Public Health Service, Department of Health, Education and Welfare. Supported in part by a grant-in-aid from the Parke Davis Company, Detroit.

[3] Professor and Head, Department of Microbiology, School of Medicine and University Hospital, University of Maryland, Baltimore, Maryland 21201.

Prompted by the existing disease problems and the favorable reception of Pakistan authorities, the University of Maryland established its present research center in Lahore in 1962. The unit has been sponsored by the Office of International Research of the Public Health Service and received important logistic support from the Government of Pakistan, the University of Maryland and the United States Agency for International Development.

The program of the University of Maryland's medical research center in Lahore is oriented towards some of those medical problems indigenous to Pakistan, such as malaria, arboviruses and arthropod-borne diseases, malnutrition, tuberculosis, enteric disorders, and other febrile illnesses. It is in the area of fevers that this discussion will focus upon primarily.

The medical setting was conducive to clinical studies and the opportunity was provided in Lahore to participate in the medical service and outpatient educational activities. Free access to interesting clinical problems was provided. Such opportunities made it possible to teach clinical investigative methods and laboratory techniques to interested students and house officers. This chance to participate in curricular programs and to appreciate other points of view was rewarding.

Young people learn medicine in Lahore's two medical schools. Steeped in the British tradition, the King Edward Medical College, established in 1860, awards the MBBS degree to men and women. The Fatima Jinnah Medical College, solely for women, was founded in 1947. Curricula follow the continental system with the baccalaureate and medical degrees awarded in five years. There are no preclinical departments of biochemistry and clinical cardiology has departmental status. Much dependence is placed upon lectures to cover basic clinical material; clinical teaching sessions begin at 0700 hours. An internal medical department consists of two clinical services each with a Professor, an Assistant Professor and a Registrar or Chief Medical Resident. A neurologist contributes to each unit. On alternate week days, one unit covers the busy medical outdoor clinic assisted by its few house surgeons. Admissions are made directly to the respective medical service. In addition to standard texts, students rely heavily on the information gleaned from lectures and at the bedside. Practical knowledge is stressed. Unfortunately, there are serious limitations in the technical laboratory support. Much reliance is placed on the careful astute elaboration of the historical features and physical findings. This is good. There is active interest in instituting full-time teaching and research programs in some of the medical schools. Pakistani physicians who have trained in England and America are available to join existing faculties provided clinical science facilities and adequate salaries can be developed.

Intimate relationships are fostered between student and faculty. One senior faculty member is a tutor for twenty or more students drawn from all five classes and periodically accompanies his group on trips to interesting areas in Pakistan. Cultural outlets and sports activities, such as cricket, hockey, tennis, field sports, and rowing, are recognized pursuits of a student's program.

Study of Febrile Diseases

A study of febrile diseases in hospital populations in Lahore was instituted by our group in order to identify systematically those diseases whose nature was not apparent at the time of initial examination. It was assumed that new agents of disease, heretofore unrecognized, would be detected, which would provide leads for a comprehensive study of their ecology. The data were expected to provide comprehensive information for the medical profession relative to the incidence and specific nature of febrile illnesses. Quantita-

TABLE I

Clinical Diagnoses of 357 Patients with Fever of Three Weeks or Less

Enteric group	
Typhoid	78
Salmonelloses	16
Dysentery	6
Respiratory	
Pneumonia, lobar	12
Pneumonia, viral	29
Viral U.R.I.	102
Meningoencephalitis	
Viral	5
Pyogenic	16
Hepatic group	
Hepatitis, viral	4
Abscess, am.	3
Viral group	
Enterovirus	11
Parotitis, orchitis	4
Variola	1
Other	42
Miscellaneous	
Malaria, leptospirosis, typhus, pharyng., hem. anemia	13
Undiagnosed	11
	357

tive data will come later through systematic observations in defined rural and urban populations. Through this study our clinical group had an excellent opportunity to combine teaching and clinical research with full access to indoor (ward) and outdoor (clinic) patients. Participation was bilateral since young Pakistani physicians were trained in study design and laboratory techniques at the center.

Patients forming the basis of the report were studied from October, 1964 to May, 1965; the laboratory data are not complete.

Results of Febrile Disease Study

Shown in Table I are the clinical diagnoses of 357 patients whose febrile illnesses were of three weeks duration or less. The initial clinical diagnosis for most patients was malaria and one or more antimalarial drugs had been administered initially. Table II provides data of 28 patients febrile for three or more weeks.

Diagnostic criteria utilized for defining the illnesses were: (1) clinical manifestations; (2) therapeutic response in some instances; (3) virologic, bacteriologic or parasitologic identification of the specific agent in the blood, stool, throat washings, other tissues; (4) serologic response; and (5) results of biopsy, such as the liver or intestine. The virologic supporting data are incomplete.

Enteric Diseases

Typhoid fever represented about 22 per cent of the short-term febrile group. The clinical patterns were variable, ranging from the classic severe type to asymptomatic forms with bacteremia. The variated clinical spectrum of typhoid, so vividly described by Osler, was encountered in Lahore; specific cases included typhoidal pneumonia, typhoidal meningitis, osteomyelitis, and myocarditis with atrial fibrillation. Six patients in the series had dual Salmonella infections, including *Salmonella typhosa* and other strains. Such dual infections create difficult diagnostic

TABLE II

Clinical Diagnoses of 28 Patients Whose Febrile Course Exceeded Three Weeks

Infectious diseases	
Tuberculosis	10
Brucellosis	1
Abscess, liver	7
Abscess, retroperitoneal	1
Cholangitis	1
Endocarditis	2
Connective tissue diseases	
S.L.E.	2
Rheumatic fever	1
Neoplastic diseases	
Lymphoma	1
Hepatoma	1
Miscellaneous	
Factitious	1
	28

and therapeutic problems; the *S. typhosa* infection responds to chloramphenicol, but there is no uniform recovery in the other salmonelloses.

Sixteen patients had Salmonella infections other than typhoid fever, including paratyphoid A and B and members of group E. There were a few patients with amoebic and bacillary dysentery.

Our clinical associates initiated a therapeutic study of chloramphenicol in typhoid patients designed to utilize less antibiotic and possibly lower the incidence of relapse. Regimens provided for fourteen continuous days of chloramphenicol and an alternate program of five days treatment, an interval of eight days without antibiotic, then five additional days of therapy. The point is unsettled, yet it appears that the interrupted course is as good as the continuous plan.

Respiratory Tract Disease

Classic lobar pneumonia which responds to penicillin and other antibiotics occurs in Lahore. There were 12 patients in the series. Throat washings and serum specimens are being assessed for predisposing causes of infection, such as influenza, respiratory syncytial, adeno, Coxsackie, psittacine viruses, *Mycoplasma pneumoniae* and *Coxiella burnetti.*

The clinical syndrome of virus pneumonia was suggested in 29 patients and approximately 100 patients experienced short, febrile, viral-like illness of the respiratory tract. Included in this latter group are approximately 25 young soldiers with headache, fever for five days, harassing cough, scattered rales and a few with a sparse morbilliform rash.

Influenza virus type A-2 has been incriminated as the specific cause in some of this group. These data are incomplete.

Meningoencephalitis Group

Five patients were encountered with either the aseptic meningitis or encephalitic clinical syndrome. Entero-viruses, mumps and lymphocytic choriomeningitis are possible causes of the aseptic types, and arthropod-borne virus diseases, such as Japanese encephalitis or dengue, are under consideration in the encephalitic group. Japanese encephalitis has been detected in Malaysia and parts of India, but not in Pakistan. Dengue virus has been isolated in Pakistan and will be mentioned later.

Pyogenic Meningitis

Pyogenic meningitis may occasionally present as obscure fever. An opportunity was provided to extend a method of therapeutic management of meningitis initiated in Baltimore in 1950. Our initial report showed that chloramphenicol is effective in meningococcal, pneumococcal and influenzal meningitis as the sole anti-microbial agent (1). The investigation was resumed in Baltimore in 1958 and the research opportunities in Lahore permitted extension of the investigation. Sixteen patients with meningitis were treated in Lahore (Table I), i.e., meningococcic—6 cases; pneumococcic—5; and undiagnosed —5. All recovered. Time will not permit a detailed analysis.

Shown in Table III are the summary data of 154 patients with various forms of purulent meningitis treated solely with chloramphenicol. The over-all fatality rate was 7.1 per cent and in the specific entities of meningococcal, pneumococcal and influenzal meningitis, the rates were 1.5, 11 and 8.5 per cent, respectively.

Our thesis is simply that multiple antibiotics are unnecessary for most cases of meningitis and these results compare favorably with the regimens of others utilizing two or more antibiotics. Such data have provided information which is of practical importance for the design of therapeutic regimens of patients with meningitis in areas of low socio-economic status. Chloramphenicol is available for oral, intravenous and intramuscular administration. It

TABLE III

Summary of Results in 154 Patients with Purulent Meningitis Treated Solely with Chloramphenicol (1950–1954; 1959–1965)

Bacterial Type	1950–54			1959–65			Totals		
	No.	Deaths	%	No.	Deaths	%	No.	Deaths	%
N. intercellularis	49	1	2	13	0	0	62	1	2
D. pneumoniae	17	3	18	19	1‡	5	36	4	11
H. influenzae	35	3	9				35	3	9
*Miscellaneous**	5	0	0	1	0	0	6	0	0
Unidentified	5	1†	20	10	2§	20	15	3	20
Total	111	8	7	43	3	7	154	11	7

* Includes one case each of *Salmonella choleraesuis*, *Listeria monocytogenes*, hemolytic *Staphylococcus*, viridans *Streptococcus*, hemolytic *Streptococcus*, and non-hemolytic *Streptococcus*. All recovered fully.

† Expired within one hour of hospitalization.

‡ Expired within four hours of hospitalization.

§ Expired within 24 and 35 hours of hospitalization.

is a major therapeutic agent for this group of ordinarily fatal cases (1).

Liver and Biliary Tract

Acute viral hepatitis is common in Lahore and several typical cases were encountered. A long-term study of convalescent patients is designed to evaluate its relationship to cirrhosis of the liver.

Patients with amoebic hepatic abscess with fever of three weeks or longer can often elude diagnosis. *Entamoeba histolytica* infection is common in tropical environments. Advantage was taken of this unique chance to study ten patients with amoebic liver abscess and to contrast their findings with patients observed in Baltimore. Patients were chosen consecutively and examined with respect to: (1) the presence of a prominence or bulge in the flank or abdomen; (2) a change in contour of intercostal spaces; (3) presence of an hepatic friction rub; (4) contour of diaphragm on lateral roentgenogram; and (5) biopsy of intact mucosa of the rectosigmoid for the presence of *E. histolytica*. The data of the 10 patients are presented in Table IV. Three patients had friction rubs over the hepatic area; each complained of pain on respiration. Seven patients had a noticeable bulge of the flank or in the anterior abdomen and one showed unmistakable evidence of fullness of the contour in the intercostal spaces in the right flank as the only pertinent exterior sign. A lateral film revealed an abnormally elevated or contoured diaphragm in six instances which was not appreciated in the anteroposterior view. The abscesses in all patients were in the right lobe and most were localized anteriorly.

Localization of infections within the liver has been attributed to two currents of blood in the portal vein, one originating from the superior mesenteric and pancreatic veins and passing into the right lobe; the other coming from the inferior mesenteric and splenic veins and passing into the left lobe (2). Ink particles injected into the splenic vein of dogs flow into the left lobe, whereas when injected into a tributary of the superior mesenteric vein, particles localize in the right lobe. Since primary foci for liver abscesses are often in those areas of the intestine drained by the superior mesenteric vein, this could explain the greater frequency of involvement of the right lobe in man, particularly in amoebic disease.

Amoebae were not demonstrated in any

TABLE IV

CLINICAL AND LABORATORY DATA OF 10 PATIENTS WITH AMOEBIC LIVER ABSCESS (PAKISTAN)

Pt.	Age	Day Dis. Adm.	Highest T. °F.	Bulge	Frict. Rub	Diag. X-Ray	Highest WBC	Aspir. Ml.	Sigmoid Biopsy	Thy. Tur.	Alk. Phos.
N.D.	20	30	101	+	0	+	12,000	N.D.	+	5.5	7.1
M.I.	50	16	104	+	+	+	14,000	N.D.	+	5.4	4.5
A.K.	30	3	103[2]	+	+	+	8,200	N.D.	—	1.7	1.5
M.Al.	25	14	103	+	0		39,800	200 (sterile)	—	3.2	—
R.A.	24	35	101	+	0	+	9,000	570	+	4.1	2.0
M.J.	25	11	103	+	+	+	22,000	240*	+	2.2	2.0
M.Ak.	24	70	103	+	0	+	4,000	600 (sterile)	—	3.6	5.5
A.B.	40	100	102[4]	0	0	+	10,000	1200* (E.H.)	0	3.3	—
G.R.	65	90	101	0	0	+	21,000	N.D.	—	4.6	9.0
A.H.	26	100	103	0	0	+	14,000	100	—	5.0	7.0

* Surgical Drainage.

of the fecal specimens; an intensive search was not made. In four patients, vegetative forms of *E. histolytica* were demonstrated in specimens of intestinal mucosa obtained by biopsy during sigmoidoscopy. Some sections taken through intact mucosa showed amoebae in the submucosa adjacent to the intact surface. (See Figure 1.) Biopsy of the rectosigmoid is a diagnostic aid in hepatic amoebic infections.

All patients were febrile. In some, the highest temperature was late at night or in the early morning which is said to typify some cases of liver abscess. Leukocytosis was usual but the count ranged from a subnormal of 4,000 to 39,800 per cu. mm. (average 14,000). Values for thymol turbidity and alkaline phosphatase were normal or only moderately elevated.

Anti-amoebic treatment was symptomatically effective promptly in all patients; one suffered four recurrent episodes and required surgical drainage in addition to other measures; the abscess contained anchovy-type exudate in which *E. histolytica* were demonstrated. The abscess of an additional patient was so large as to require surgical drainage after failure of anti-amoebic drugs and aspiration. All patients recovered, four with medical treatment alone, four with combined medical treatment and aspiration of the liver abscess, and two with medical treatment, aspiration and surgical drainage. Striking in all of these patients was the lack of toxicity or the presence of nausea and vomiting which characterize peritonitis and other intra-abdominal inflammatory conditions.

Table V presents the data of 12 patients with liver abscess hospitalized in Baltimore from 1958 to 1964. Two of four patients with amoebic abscess died, and one patient with a secondarily infected amoebic abscess died. Two of six patients with pyogenic abscess of the liver died. Pertinently, the fatal cases of amoebic abscess were institutionalized psychotic patients who were admitted with far-advanced disease. In 11 instances, the abscesses were in the right lobe and multiple and bilateral in one fatal case. The etiology of one case was undetermined. Excluding the patient with multiple abscesses, the roentgenograms were reported as diagnostically helpful in only six of eleven cases. A review revealed that some of these films should have been reported as abnormal. All patients showed a leukocytosis ranging from 11,600 to 53,000 per cu. mm. (aver-

FIG. 1. *Entamoeba histolytica* in section of intact rectosigmoid mucosa obtained by biopsy. Note Trophozoites, with central karyosomes, in capillaries.

age 21,000). Two patients showed moderate bilirubinemia, 8.5 and 5.5 mg./100 cc., and one a high value of 55 mg./100 cc. Five patients showed elevation of the thymol turbidity reaction and eight showed elevated values of alkaline phosphatase (Bodansky units). Complications occurred in eight patients, such as empyema, subdiaphragmatic or subhepatic abscess, peritonitis, pericarditis, and pyelophlebitis. Four of eight patients with serious complications recovered, three with antiamoebic

TABLE V

CLINICAL AND LABORATORY DATA OF 12 PATIENTS WITH AMOEBIC AND PYOGENIC ABSCESSES (BALTIMORE)

Age	D. D. Adm.	High. T. °F.	Etiology	Location	Diag. X-Ray	WBC	Vd. B.	Thy. Tur.	Alk. Phos.	Ther.	Complicat.	Result
51	120	103	Am.	U.R.	Yes	14.1	1.5	4.0	8.0	C.Q.*	Empy.	D
8	16	105	Am.	U.R.	No	28.0	2.6	12.3	7.3	Em.	S.D.A., peric.	D
47	180	102[2]	Am., Staph.	L.R.	No	20.0	2.6	9.0	6.6	Abs.	Periton.	D
25	14	104	E. coli, Bacteroides, Staph.	U.R.A.	Yes	11.6	8.5	4.4	7.4	Abs.*	Periton.	D
46	80	103[6]	Am.	R.	Yes	17.3	1.5	13.4	10.8	Em. C.Q.	None	R
24	30	102	Py.	R.A.	No	19.1	1.3	2.6	1.7	Em.* tm.	S.H.A.	R
58	30	103[6]	Py.	R.L.	Yes	19.7	5.0	8.2	4.3	Pen.* cm.	S.D.A.	R
66	8	101[4]	Alpha strep.	U.R.A.	No	35.3	.8	2.0	4.4	—	Periton.	R
44	60	104	Beta strep.	U.R.P.	Yes	17.5	1.4	8.0	5.7	Pen. sm.	None	R
61	Unk.	101[8]	Staph.	Bilat.	No		.9	1.8	2.9	—	Ulcer, fistula, pyleph.	D
19	11	104	Am.	U.R.	No	21.3	2.7	1.5	11.9	Em.* C.Q.	None	R
27	17	100	Unk.	U.R.	Yes	53.0	35.0	3.0	19.8	Abs.*	Shock	R

No instances of Bulge, Friction Rub, Biopsy Sigmoid.
* Surgical Drainage.

treatment and surgical drainage, and one with medical treatment alone. In none of the hospital records was the presence or absence of an external bulge or prominence, friction rub or rectosigmoid biopsy mentioned.

Pertinent summary data of the two series are shown in Table VI. Obviously, the continued exposure to, and familiarity with, a disease is germane to prompt diagnosis and application of appropriate curative methods. Simple bedside observations are of great value.

TABLE VI

SUMMARY DATA OF 22 PATIENTS WITH HEPATIC ABSCESS

The lesions of 21 patients were in the right lobe.

Type	Maryland	Pakistan	Right Lobe	Average WBC	Surgical Drainage	Number Recovered
Amoebic	6	10	16	14,000	5	13/16
Pyogenic	5*	0	4	21,000	2	3/5
Sterile	1		1	53,000	1	1/1

* 1 Fatal case; bilateral multiple abscesses.

Suspected Viral Group

Forty-two patients were thought to have a viral disease based on the clustering of cases, a febrile course of three to five days without localizing signs, normal or low blood leukocyte counts, negative malaria smears, relative bradycardia and a mild course.

Our group was successful in isolating dengue virus from patients with a short-term, febrile illness known as Dacca fever in East Pakistan (3). The agent has not been recovered in the Western sector although serologic and virologic studies are incomplete. A short, febrile disease resembling dengue, associated with headache, malaise and a morbilliform rash, has been specifically related to West Nile virus infection.

ECHO or Coxsackie virus infections were suggested in ten patients; classic para-

lytic poliomyelitis was encountered once. Several cases of parotitis and orchitis suggested mumps infection although Coxsackie virus disease must be excluded. One patient masqueraded as fever without localizing signs for several days until the characteristic rash of variola developed. Backache was *not* present and the initial lesions resembled rose spots.

Miscellaneous Causes of Fever

Malaria: Although the majority of the 385 febrile patients were regarded initially as having malaria and treated with antimalarial drugs, only four were confirmed as having the disease; they recovered.

Typhus Fever: Classic murine typhus in a soldier was confirmed by demonstration of a rising and diagnostic titer of agglutinins for Proteus OX-19 and complement-fixing antibody.

Hemolytic Anemia: Several febrile patients were treated initially with primaquine or quinacrine for malaria and one patient received sulfone tablets for a skin lesion. These patients were later called to our attention because of fever, dark urine (hemoglobinuria) and severe anemia, each having hemolyzed about 80 per cent of their erythrocytes. All four patients recovered after supportive care and cessation of drug treatment. Important is the fact that none of the patients with hemolytic anemia required the drugs which produced their very serious illnesses. Each patient and members of their families showed a deficiency of glucose 6-phosphate dehydrogenase in their erythrocytes (4). This inherited trait, carried in the X chromosome, has been reported in Africa, America, the Mediterranean area, and the Far and Middle East, although not in Lahore. A limited survey of approximately 500 students and military personnel in West Pakistan revealed an incidence varying from 1 to 3 per cent.

Febrile Diseases Exceeding Three Weeks

Twenty-eight patients were encountered whose febrile courses exceeded three weeks. The specific causes varied and are comparable to those encountered in other environments. Infectious diseases represented the highest incidence. A few patients with bacterial endocarditis, hepatic abscess, cholangitis, brucellosis and tuberculosis were studied. Non-infectious causes were rheumatic fever, systemic lupus erythematosus, hepatoma and lymphoma.

Tuberculosis is widespread in West Pakistan and is a common cause of fever. It often eludes diagnosis and mimics other diseases since the clinical signs are not always obvious. The nature of the febrile illness in the first 10 patients, cited in Table II, was obscure initially. After appraisal of the clinical manifestations, the febrile course and hepatic biopsy, seven were shown to have granulomatous lesions of the liver regarded as tuberculous. None of these patients showed obvious pulmonary foci. Hepatomegaly was present in each and five showed splenomegaly. One of the patients, whose diagnosis was unmistakable, died in spite of intensive anti-tuberculous therapy for six weeks.

The concept that tuberculosis involves the liver without obvious pulmonary manifestations was new to this environment. Clinicians found this information useful since tuberculosis is a relatively common cause of the obscure fever syndrome which can be identified by hepatic biopsy.

Brucellosis was not encountered as commonly as anticipated.

General

The program of Maryland's research center in Lahore is broad and permits unique opportunities for all types of clinical experience. There are chances to perform cursory examinations in villages to determine spleen indices in support of the

malarial study program. The splenic indices and rates of parasitemia were 40 per cent in some areas.

In spite of abject poverty and malnutrition in villages, people are affable, clean and cooperative. Bronchitis and emphysema were common in older adults and there were constant requests for help to palliate coughing and wheezing. Men practice the ancient habit of hookah smoking which they begin as boys. Smoke travels from the charcoal and tobacco through several feet of tubing, filtration through water, and inhalation. Although postmortem examinations are seldom performed in Pakistan, the advent of chest surgery has shown that bronchogenic carcinoma is prevalent. Determination of its incidence in hookah smokers might be pertinent since the element of heat and the effect of toxic products from cigarette paper are removed.

Another interesting village experience was provided by observing cases of smallpox in urban and rural settings. The new anti-viral drug, Marboran, was tested for its chemoprophylactic value in contact subjects and also for its therapeutic efficacy. It prevents the disease in contacts, which was shown initially in Madras, but appears to have little therapeutic value.

In summary, rewards accrue through our participation in clinical, educational and investigative programs overseas. Global medical problems are not unique to any country and the new knowledge gained profits both sides. By reaching beyond the confines of the United States, we avoid fostering our immaturity and ignorance of the problems of others.

A qualitative study of febrile diseases in Lahore hospitals yielded a wide spectrum of entities, some of which are newly described.

1. Typhoid fever masquerades in many forms. It occurs in cool weather as well as summer and represents approximately 20 per cent of obscure fevers. It responds to chloramphenicol therapy uniformly and anti-bacterial resistance of *S. typhosa* is uncommon.

2. Dual Salmonella infections occur simultaneously and can confuse the clinical picture with respect to response to treatment.

3. Short-term viral infections of the upper and lower respiratory tract play a significant role as a cause of fever. Influenza virus infection, type A-2, has been identified.

4. Based on clinical evidence, viral encephalitis occurs primarily in children in Lahore. Arthropod-borne agents are suspected.

5. Pneumococci and meningococci are common causes of pyogenic meningitis and yield to chloramphenicol as the sole antimicrobial drug.

6. Viral hepatitis occurs in its characteristic clinical forms; its relationship to the later development of cirrhosis is unclear.

7. Amoebic liver abscess is common in Lahore; helpful bedside clues to localization are: (1) an obvious bulge of the chest wall and abdomen or prominence of intercostal interspaces; (2) presence of an hepatic friction rub; and (3) interpretation of the diaphragmatic contour in the lateral roentgen view. Biopsy of intact rectosigmoid mucosa may reveal amoebae when fecal specimens are negative.

8. Short-term viral infections occur in Pakistan. Dengue b and West Nile viruses have been identified.

9. Malaria occurred uncommonly as a cause of febrile illness in Lahore during the months of October, 1964 to May, 1965.

10. Deficiency of the enzyme, glucose-6-phosphate dehydrogenase, is related to the pathogenesis of hemolytic anemia in Pakistan; the general incidence of the genetic defect is approximately 2 per cent.

11. Tuberculosis of the liver and other abdominal organs is a relatively common

cause of the obscure fever syndrome in Lahore.

Possibly some of these findings will provide diagnostic and therapeutic guidelines useful to the medical practitioner.

During this sabbatical leave, it was not possible to escape administrative chores, including an exercise that any agency must encounter that uses public funds; reference is made to a "site visit." One must skillfully avoid something known as being "phased out!" These experiences were pleasant and they really proved more tiring for the visitor than the visited. Educators and investigators now travel widely around the world and we were delighted that Lahore, the Kingdom of Swat, and the Khyber Pass attracted many American scientists to this interesting territory. News was always exchanged avidly and visiting teachers enjoyed giving lectures or conducting traditional teaching clinics with students. It was a heavy responsibility to insure that one's friends visited the proper monuments, had enough film, made wise purchases, avoided the local quickstep, and made the airplane.

Acknowledgments

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References

1. Parker, R. T., Snyder, M. J., Liu, R. S., Looper, J. W., Jr., and Woodward, T. E.: Therapeutic range of chloramphenicol in purulent meningitis. Antibiotic Med. 1: 191, 1955.
2. Serege, H.: Etude sur l'independence anatomique et physiologique des lobes du foie. J. de Med. de Bordeaux 32: 327, 341, 357, 1902.
3. Russell, P. K., Buescher, E. L., McCown, J. M., and Ordonez, J.: Recovery of dengue viruses from patients during epidemics in Puerto Rico and East Pakistan. Submitted for publication.
4. Ronald, A. R., Underwood, B. A., Hasan, S., Basir, M. A., Gallager, L. R., Fatima, N., Khan, M., Mallick, S. M. K., and Woodward, T. E.: Primaquine sensitive haemolytic anemia in West Pakistan. Pakistan J. Med. Res. 5: 5, 1966.

Skin Carcinogenesis as a Laboratory and Clinical Model in Cancer Research[1]

By EUGENE J. VAN SCOTT, M.D.[2]

I am pleased and honored to give the third Harry Shay Memorial Lecture, a memorial tribute to Dr. Shay, who as a gastroenterologist was motivational in the establishment of the Fels Research Institute and the development of its program on cancer research.

It is said that politics makes strange bedfellows, and strange bedfellows make interesting politics. There are equivalents to this in cancer research where oncology leads to cross fertilization of scientific disciplines, and cross fertilization makes very interesting cancer science. Cross fertilization of scientific disciplines evolves in diverse ways, the logic of evolution being often seen only in retrospect. I am reminded in this regard of Dr. John R. Heller, who became Director of the National Cancer Institute after World War II during which time he had been Chief of the Venereal Diseases Division of the Public Health Service. The program of the V.D. Division, an intensive treatment attack on syphilis, all but entirely eradicated syphilis from dermatology clinics in this country by the end of the war years. Asked by what reason of logic did he, a syphilologist, turn his attention to oncology, Dr. Heller has recorded the classic answer that after they had knocked the "H" out of "chancre", this left them with "cancer". So you see there are many threads of relationships in the development of cancer research!

[1] Harry Shay Memorial Lecture III, Temple University School of Medicine, 16 March 1966. Published in honor of Dr. Shay, late Fellow of The College of Physicians of Philadelphia and a co-recipient of its Alvarenga Prize.

[2] Director of Intramural Research, National Cancer Institute, National Institutes of Health, Bethesda, Maryland 20014.

It seems to me fitting that the Skin and Cancer Hospital and the Fels Research Institute are associated in co-sponsoring this symposium today on skin carcinogenesis, and have incorporated the Harry Shay Lecture into the program. As a gastroenterologist, Dr. Shay involved himself with the internal integument, which indeed is joined with the external integument. Moreover, Dr. Shay and his associates further strengthened this bond by demonstrating that instillation of methylcholanthrene into the stomach of rats lead to cancer of the breast, an organ which is a cutaneous appendage.

Modern oncology begins with the skin. Dr. Michael B. Shimkin recently recorded this by his perceptive selection of the initial cover illustration for the journal, *Cancer Research,* a picture of Percivall Pott, and that of a chimney sweep and his helper. Thus almost 200 years ago the initial example of environmental carcinogenesis was identified in the skin. The choice of the next cover illustration of *Cancer Research* again signaled the role of skin in studies of carcinogenesis, by the photos of Yamagiwa and Ichikawa, the Japanese workers who demonstrated the causal role of tar products and cancer on the ears of rabbits.

Why has the skin been so involved in carcinogenesis, particularly environmental carcinogenesis? The reason is that each biologic package is contained in an integumental wrapper, which is the interface between the organism and the external environment. For the unicellular organism all communication, as far as is known, is by direct transmission through its membranous integument. Multicellular organisms have developed more complex con-

tainers, whereby instead of ingesting materials of the environment by direct contact-absorbance or engulfment, the environment is shunted into interior canals for usually more selective assimilation, through the processes of digestion or respiration.

Phylogenetic developments would have had little to do with environmental carcinogenesis had not the environment contained carcinogens. Things were not bad enough with actinic radiations, but man discovered fire and proceeded to create artificial environments, atmospheric and otherwise. Whereas the primitive organism was able to recognize certain noxious items in its environment, and to reject them because of texture, temperature or taste characteristics, sensory mechanisms did not evolve to keep pace with man's synthetic adventures. Hence the sensation of the warmth of the chimney gave no hint to the chimney sweep concerning the penetration of tar into his scrotal epidermis. Nor was the internal integument spared such trickery, for the exhilarating pulmonic sensation of cigarette smoke inhalation carried with it no warning tickle of the volatilized carcinogen. And gustatory satisfaction of charred foods has no unpleasantness to identify possible carcinogens.

The skin, at least up to the present, has borne the brunt of man's misadventures with his carcinogenic surroundings, as is indicated by the high incidence of skin cancer. Next in order in our system of integuments is the environmental carcinogenic burden of the pulmonary epithelium. And in third place is gastrointestinal epithelium, which has the lower frequency of cancer. This order I suppose indicates that man is more discerning of what he eats than what he smells or touches.

To turn now to the title of this lecture, namely "Skin Carcinogenesis as a Laboratory and Clinical Model in Cancer Research", it seems useful to define some of the terms therein. If we are to address ourselves to the origin and process of development of cancer, that is, "carcinogenesis", it is important to emphasize that it is in fact a process, perhaps a multi-staged process. A definition of "cancer" is impossible to formulate adequately and I am sure we can quietly agree that cancer research is still in the embarrassing position of not knowing precisely what it is trying to treat or prevent.

The skin is very instructive in a consideration of carcinogenesis as a process; and the numerous cancerous and cancer-like conditions identifiable in the skin highlight the question of what should or should not be called cancer. In this atmosphere of inquiry, the skin stands out as a model since it presents its questions quite visibly. Other qualifications can be added. For example, in the skin all etiologic categorizations of cancer are found, that is, physical, chemical, viral, genetic and immunocarcinogenesis. Such lists, however, are attributes of limited value to us in an understanding of cancer and carcinogenesis and I would choose to otherwise illustrate the skin as a model in cancer research, through a consideration of cancer in two cell systems: the epithelial cell system and the lymphoreticular cell system.

Characterization of an abnormal thing or event depends upon its comparison to normal. Much of what we identify as cancer is its behavior, what it does to the host. A cancer cell is to the whole organism much as the criminal is to society, where both are known by the deed committed. But whereas the criminal is capable of normal behavior, we tend to lead ourselves into thinking the cancer cell is capable of only abnormal behavior. This would not seem to be true, for on the basis of projections from what we know of biologic events in general, that there are *ranges* of expression. Hence, it seems reasonable to think of degrees of cancer, the degree of a cell's departure from normal. For this we must first know the ranges of normal capa-

bilities of the cell, and for convenience and illustration these may be grouped into 1. mitotic and synthetic potentials, and 2. response to external controls.

The range of mitotic expression of the integumental epithelial cell is enormous, varying from a practically zero level in the epithelial sac of the dormant, telogen, hair root, to the high rates found in germinative hair matrix cells where mitotic indices approaching 10% are found and doubling times of less than 13 hours (5). Between these extremes are intermediate rates, for example of normal epidermis where the turnover time of the germinative basal cells is 5–6 days (2). Suffice to say in this cursory comparison that the mitotic expression of the cancer cell falls within the range of normal. The range of synthetic potential of the integumental epithelial cell is similarly wide. The cell has inherent capabilities of synthesizing different fibrous protein depending upon whether it is located in the epidermis, the oral mucosa, or in the hair root. In other locations it may produce lipids, as in the sebaceous gland, or globular proteins, as in the apocrine gland.

What is important to realize is that a single cell seems to possess all these capabilities, and the actual synthetic product is a function of the cell's anatomic location in the integument. A few examples may be used to illustrate this. One example is found in wounds of the skin either accidentally or experimentally inflicted. In such circumstances cells participating in the repair process may assume functions different from those performed before the injury. For instance, sebaceous gland cells may move to the skin surface, re-epithelialize a denuded area, and remain there and function as normal epidermal cells. Indeed, in the yearly-renewed deer antler surface epidermal cells give rise to all epithelial components of skin (1). Other examples are found in transplantation studies which have shown pure oral mucosa, transplanted sub-epidermally to the skin of the back, keratinizes with a granular layer in a fashion characteristic of skin epidermis (6).

The integumental epithelial cell not only is responsive in these ways to environmental influences within the skin, but its survival alone seems dependent upon the skin. This conclusion can be derived from the observation that the majority of injection needles are found to contain epithelial fragments after introduction through the skin (3). Thus skin epithelium is frequently injected sub-cutaneously and intravenously, but no evidence of further survival of these injected fragments is known.

Comparison of the foregoing mitotic, synthetic, and response attributes of the normal integumental epithelial cell with attributes of cells of epithelial skin cancers leads one to a tentative conclusion that at least some lesions we know as cancer are comprised of cells showing at best, minimal departures from the normal. An example would be cells of the basal cell carcinoma, a lesion which as you know is a non-metastasizing cancer, seemingly dependent upon residence within the skin for its survival. And in fact, studies in which bits of pure tumor have been implanted subcutaneously in man have shown this dependence to exist (6). The tumor is successfully transplanted to subcutaneous sites only if its native surrounding connective tissue from the dermis is transplanted with the tumor fragment. Further biochemical and electron microscopic comparisons of the cells of a basal cell tumor lead one to conclude that the cell is characterized by a single defect, a defect which prevents the cell from fulfilling its synthetic function, the production of fibrous protein keratin which is required for the process of keratinization and cell death (4).

Thus the basal cell tumor cell, departing very little from the normal, may be comparable to the rather benign but so-called cancerous lesion experimentally in-

duced in the liver of rats by low-dose feeding of carcinogens. Such tumors, the Morris minimal deviation hepatomas, have been the subject of extensive study by Dr. Sidney Weinhouse here at the Fels Institute. I must concede these tumors may be as provocatively interesting as the basal cell tumor.

Characterization of cells from other skin epithelial tumors is not yet precisely possible since appropriate studies have yet to be carried out. But from what is casually known of cells of a squamous cell cancer, it seems clear that the degree of departure from normal is considerably greater than that found for the basal cell cancer.

I believe the foregoing illustrates that useful approaches to a definition of cancer can be made through the skin and its various lesions. And these various lesions, in fact, provoke our attention toward what to me is a most engaging phenomena, that of the reversibility of the carcinogenic process and the reversibility of cancer.

For the sake of continuity we will begin this subject by calling attention to an epithelial lesion now known as keratoacanthoma, a lesion first introduced under the term "self-healing" squamous cell cancer. And as far as we know today, the lesion is just that. Although the histologic features of the lesion are characteristic enough to be identified as one that usually regresses without treatment, such characteristic lesions are known also to pursue a decidedly malignant course. Spontaneous disappearance of keratoacanthoma is associated with a heavy local infiltration of inflammatory cells, but it has not yet been demonstrated whether this is or is not an indication of autoimmune recognition and rejection.

Another example of spontaneous reversibility or control of epithelial neoplasia is encountered in both laboratory animals and in man. In laboratory animals such a lesion is the cutaneous papilloma that develops in response to topical applications of a chemical carcinogen. In man such a lesion is the actinic keratosis that appears in skin exposed to years of sunlight. In both animals and man such lesions are known to progress further into invasive or metastasizing epithelial cancers. But for some reason we have failed to focus attention on the fact that the vast majority of these precancerous lesions do not progress into something we would call cancer, but either remain in an equilibrium state in the skin, or disappear. Although the frequency of disappearance of actinic keratoses from the skin of man has not been determined, casual clinical observations suggest this event may be quite usual.

In the time remaining I would like to divert your attention from cutaneous epithelial carcinogenesis to the genesis and course of *lymphoma* in the skin. The characteristic cutaneous lymphoma of man retains the inappropriate name mycosis fungoides. The initiation of the disease frequently is associated with an allergic event in the skin, clinically diagnosed as an eczema of unknown etiology, a contact allergic dermatitis of known cause, or not infrequently as an allergic cutaneous eruption due to a systemically administered drug. Certain histologic features identify the early disease, and for the most part the infiltrate consists of lympho-reticular cells, benign in appearance. Further development is accompanied by an ever increasing infiltration of the skin by lympho-reticular cells appearing more and more morphologically abnormal. The course of the disease is known to be a fatal one in which the lymphomatous process, initiated in the skin, progresses to systemic involvement.

As with epithelial precancers, we have tended to ignore those cases of early mycosis fungoides which tend to remain in equilibrium with the host. And those cases in which the early disease disappears have been regarded as evidence that mycosis fungoides is really not a cancerous disease to begin with. I believe this circular logic has kept us from making important ob-

servations on the nature of this lymphoma's reversibility. Instead of inviting skepticism which might surround considerations of the early disease, I would like to relate to you some interesting occurrences occasionally encountered in the fully developed disease.

One event is that cutaneous lesions, although frequently initiated by what seems to be an allergic episode, can be made to disappear by a contact allergic dermatitis induced by one of several sensitizing agents (unpublished data). Another is that cutaneous lesions of some patients have a tendency to spontaneously disappear rather than progress. And finally, we have observed a patient with far advanced disease who experienced a complete disappearance of the disease following an allergic drug reaction (personal observation, to be published).

Hence, in the skin we encounter what may be another model for study of carcinogenesis, the unusual model of a lymphoma, a cancer with immunologic determinants in its genesis and immunologic determinants in its course. There are good reasons for extending our interests in this model as well as the model provided by epithelial carcinogenesis.

In summary, my thesis of this afternoon would include the proposition that carcinogenic events in the skin can be useful as a study model in cancer research. That past research can be emulated, but research on cutaneous carcinogenesis is to be extended into areas heretofore neglected. And seeming to be particularly exploitable for study in the skin is the phenomenon of the reversibility of cancer processes, of which the skin demonstrates several models.

REFERENCES

1. Billingham, R. E., Mangold, R., and Silvers, W. K.: The Neogenesis of Skin in the Antlers of Deer. Ann. N.Y. Acad. Sci. 83: 491–498, 1959.
2. Epstein, W. L., and Maibach, H. I.: Cell Renewal in Human Epidermis. Arch. Derm. 92: 462–468, 1965.
3. Little, D. C.: Skin Fragments in End-Opening Needles. Canad. Med. Ass. J. 72: 374–375, 1955.
4. Van Scott, E. J.: Definition of Epidermal Cancer. In: W. Montagna and W. C. Lobitz (Eds.), *The Epidermis*. pp. 573–586. New York, Academic Press, Inc., 1964.
5. Van Scott, E. J., Ekel, T. M., and Auerbach, R.: Determinants of Rate and Kinetics of Cell Division in Scalp Hair. J. Invest. Derm. 41: 269–273, 1963.
6. Van Scott, E. J., and Reinertson, R. P.: The Modulating Influence of Stromal Environment on Epithelial Cells Studied in Human Autotransplants. J. Invest. Derm. 36: 109–131, 1961.

James Hutchinson (1752–1793): Letters from an American Student in London[1]

By WHITFIELD J. BELL, Jr., Ph.D.[2]

THE story of the young Americans who went to Europe to study medicine after the middle of the 18th century has been often told. Very much aware of the significance of what they were doing, they travelled to London, Edinburgh, and Paris to train themselves for practice and for teaching; and they returned to establish standards for both. Thomas Bond studied in London, Edinburgh, and Paris and had his degree from Rheims; Benjamin Morris and John Redman were graduated at Leyden; the members of the first faculty of the Medical School of the University of Pennsylvania—John Morgan, William Shippen Jr., Adam Kuhn, and Benjamin Rush—were all graduates of Edinburgh. The example they set was followed by their students—Thomas Parke, Samuel P. Griffitts, Caspar Wistar, George Logan, and others far less well known. One of these was James Hutchinson.[3]

Well enough known in his own day, Hutchinson was for 14 years a physician of the Pennsylvania Hospital, for 11 years one of the secretaries of the American Philosophical Society, and a founder and first secretary of the College of Physicians of Philadelphia. After the Revolution he took a prominent part in politics; and his figure on the streets—for he was a man of gigantic girth—must have been one of the familiar sights of the city. But he never wrote anything over his own name; he died untimely in the first days of the yellow fever epidemic of 1793, when his loss went almost unnoticed among infinitely numerous similar losses; and so he has become little more than a name in the 20th century.

Born in Bucks County, Pa., in 1752 and educated in local schools, Hutchinson was apprenticed first to the apothecaries Isaac and Moses Bartram, from whom he acquired "no mean proficiency in Botany, Chemistry, & Pharmacy," and then to Dr. Cadwalader Evans, a successful Quaker physician who was a member of the staff of the Pennsylvania Hospital. In 1771 he entered the Medical School of the College of Philadelphia, and was graduated three years later with the degree of bachelor of medicine and a gold medal specially awarded by the Trustees for excellence in chemistry. Meanwhile, with recommendations from his professor Dr. Benjamin Rush and his preceptors Dr. Evans and Isaac Bartram (who judged him "fully capable of conducting any one of the Processes in the Pharmaceutical Parts of Chymistry"),[4] Hutchinson had been named apothecary at the Hospital. He held the post two years.

Thus by 1775 Hutchinson had had about as much, and as good, formal training and experience in medicine as America could offer a student; and he could have settled in Philadelphia or a neighboring town and practiced with credit and profit. But

[1] A portion, revised and enlarged, of the Kate Hurd Mead Lecture XVII, The College of Physicians of Philadelphia, 26 April 1966. A fuller account of Hutchinson as physician and politician will be published in the near future.

[2] Librarian, The American Philosophical Society, Philadelphia, Pennsylvania 19106.

[3] For biographical sketches, see James Thacher, *American Medical Biography* (Boston, 1828), I, 309–11; University of Pennsylvania *Alumni Register*, IV, no. 3 (mid-March 1900), 8–10; *Dictionary of American Biography*.

[4] Isaac Bartram, Certificate, [1773]; Cadwalader Evans, Certificate, April 19, 1773, Hutchinson Papers, American Philosophical Society.

Fig. 1. Portrait by G. P. A. Healy at the University of Pennsylvania School of Medicine, from a miniature by Charles Willson Peale.

Hutchinson entertained higher ambitions, and, by a curious twist, the political situation put him on the way to achieving them.

Hutchinson was a Quaker, who knew that he was supposed to be peaceful, neutral, and loyal. But the events of the winter of 1774–75—the first Continental Congress, the Non-Importation Agreement, the closing of the port of Boston, military preparations everywhere—excited him in most un-Friendly ways. These reactions were observed with mounting concern by Hutchinson's uncle, the wealthy merchant Israel Pemberton, one of the weightiest Friends in the Yearly Meeting. Accordingly, to remove the young man from the unsettling environment and also to further his education, Pemberton offered to send his nephew to England for a year's study.

Hutchinson was well prepared by study and practical experience to take advantage of London's opportunities for medical study. In addition he carried the best introductions to members of the sober, prosperous, influential Quaker society of London, especially to Dr. John Fothergill, friend and correspondent of Israel Pemberton,

one of the city's busiest and best known practitioners, who took a deep interest in anyone from Pennsylvania. One of his letters was to a young Quaker physician Dr. John Coakley Lettsom, who introduced Hutchinson at once to the practice of Aldersgate Infirmary.[5]

Most American medical students in Britain during the preceding 15 years had paid little attention to surgery. Some doubtless had a strong revulsion against its horrors; but many had scorned it as inferior to medicine. Fothergill had little patience with such notions. He strongly urged Hutchinson to prepare for a career in surgery, and he advised him to register as a pupil of Percivall Pott at St. Bartholomew's Hospital. Though Pott's 50-guinea fee appalled him, Hutchinson enrolled at Bart's on October 3, 1775, and fell to work. He also registered as a pupil of Dr. William Hunter in anatomy and of Hunter's brother John in surgery. To his uncle Pemberton, Hutchinson reported what he was doing and confided his hope that he might become a teacher in Philadelphia, like Bond and Morgan and Shippen. Nor could he keep political news and speculation from his mind; and, though he knew his sentiments were sure to distress his uncle, he could not keep his thoughts from spilling from his pen.[6]

James Hutchinson to Israel Pemberton

London, 20th 12mo 1775

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When the October packet sailed we were told that it would be the last that would be sent to America, therefore I wrote thee fully by it, and gave thee a particular account of my situation, Plan, my expences, and Prospects. Since then I have pursued my Studies with unremitting diligence, have been advised and directed in all my measures by my kind Friend D^{r} Fothergil, have continued pursuing the same course I mentioned in my letter by the Packet, with only this alteration, that I attend another Course of Lectures on Surgery in the evening, which are delivered by John Hunter, Brother to D^{r} William Hunter; these add five Guineas to my expences.—

I have the greatest Prospects of improvement in Surgery, I have great Opportunities, and great advantages. The Professors take me by the Hand, and the Surgeons of eminence are all kind to me. I dress daily at the Hospital, and am often permitted to perform some of the less Capital Operations myself, and generally assist in the performing of the more Capital ones; this distinction I owe in a great degree to the kindness of Surgeon Pott;—in fine should my endeavours be blessed by providence with success, and should I live to return to my native land, I am flattered by my friends that I shall sometime be capable of delivering a Course of Lectures on this very important Branch of Medicine. This at present however is only *Ideal*, and I would not wish my *Medical Friends* in Philadelphia to know that I had even an *Idea* of such a thing. A person capable of this is much wanted in Philadelphia; for such there is a good vacancy, and as I am so happy as to stand well with the Pupils, there will be no harm in endeavouring to be qualified for such an undertaking.

But tho' my attention is principally directed to Surgery and Midwifery, yet I by no means neglect Physic. I wish for improvement in every part of the Healing Art; I wish to be able to expel the raging Fever, to make diseases die.—But why do I indulge thoughts of this kind, when by the present appearance of things it will be difficult, or perhaps impossible to return, as it is generally imagined by people well acquainted with public Affairs, that there will be no Vessels from hence to America next season except on Governments account. Reflections that these things cause to arise, prevent me from being as easy as I could wish, and in some measure interrupt my Studies. I have too great an affection for my Country not to feel for its distresses; its dearness to me causes me to share in the troubles, partake of the Calamities, and sympathise with the afflictions that now surround it. It is my constant wish & prayer that a reconciliation may take place, but it should be a constitutional one; any other would be only trifling and give rise to future disputes. Oh! that the voice of Justice and reason could but be heard; they would have a much better effect than the mouth of the Cannon, or the Point of the Bayonet. I shudder at the thoughts of the Blood that will be spilt next summer, and strive as much as possible to prevent the dreadful, the Horrid Idea from rushing into my mind, but in Vain. I must however avoid saying anything on Politics, otherwise I shall plunge myself in a Labyrinth from which I could not with ease extricate myself. I could inform thee of the Situation of Public Affairs, but it would be dangerous, it would be imprudent, and Alas! they all seem to partake of that kind that would give my dear Uncle pain.

[5] Lettsom to Thomas Parke, September 10, 1775, Hutchinson Papers.

[6] This and other letters quoted in this article are in the Hutchinson Papers.

A few months later Hutchinson wrote Pemberton again. He repeated some of what he had written before (because he could not be sure his letters would arrive), but also spoke of his professors' excellencies and the reasons why London was a better place than Philadelphia to study practical surgery. To hospital attendance with Pott and the lectures and demonstrations of the Hunters, Hutchinson now added courses in chemistry, materia medica, and practice taught at three successive hours each morning from 7 to 10 o'clock by Dr. George Fordyce, the popular and tireless physician to St. Thomas's Hospital. He also bought tickets for the midwifery lectures of Dr. Thomas Denman, physician and accoucheur of Middlesex Hospital, and for the botany lectures of Dr. William Curtis, an agreeable young Quaker. In consequence Hutchinson had the privilege of observing practice at most of the principal hospitals. He was getting a kind of surgical training none of his predecessors had had.

James Hutchinson to Israel Pemberton

London April 7th 1776

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With regard to my Studies I go on pursuing the plan I have so often acquainted thee I had adopted. My Winter courses are now nearly all finished. Dr Hunter's Lectures will be concluded in about three weeks, and John Hunter's & Percival Potts' in about four weeks. I have been a Dresser at the Hospital seven months. My Plan has been very different from any of my Predecessors. While in Philadelphia my attention was principally, indeed almost wholly devoted to Physic, & without flattering my Alma Mater, I think I can say, the Science of Medicine is better taught in the Colledge of Philadelphia than in London; but the Case is very different with respect to Surgery. Those persons who have studied in Philadelphia and received instruction from no other quarter, cannot possibly be good Surgeons; the want of Cases, the want of Objects; and last of all the want of teachers, must effectually put it out of their power. To that branch of the healing Art my attention has as yet been entirely devoted. My Countrymen that have been over here before me, have applied to Physic in General; few of them have ever attended a regular course of Lectures on Surgery, and still fewer have made it their principal Object. My own inclination and the advice of my friends, induced me to consider Surgery, as the Science in which I am to expect the greatest improvement, while in England; and of consequence I have spared no pains to make myself acquainted with it, and I am happy in saying thro' the blessing of providence, I have every opportunity of acquiring knowledge on this subject. I am favoured with many good friends amongst those who are capable of giving instruction; they treat me kindly, are free in communicating useful knowledge; & I am willing and anxious to receive it. Tho' I am a pupil to Bartholomew's Hospital only, yet there are few curious cases, that are worthy of particular Observation, in the other Hospitals but what I have an Opportunity of seeing, and there are but few capital Operations performed, that I am not invited to. From Potts', and from John Hunter's Lectures, I have received great advantage; the first of these is eminent for his Judicious Practical Observations, and the latter for his Theoretic knowledge, and his indefatigable industry in investigating the hitherto concealed laws of the Animal Oeconomy. His Brother Dr Hunter is probably the best Anatomist now living, and he is perhaps the best Physiologist.

Notwithstanding what I have said above concerning Surgery, I shall not neglect Physic. Tho' my time has as yet been wholly taken up with dressing, dissecting, Anatomical & Chirurgical Lectures, yet those courses are now nearly over, and in a few days I shall enter with Fordyce on the Materia Medica, Chemistry, and the Theory & Practice of Physic; these will occupy the morning; while Midwifery Lectures will engage me in the afternoon. These will be all concluded in between four & five months, when I shall be ready to return, if I should be so favoured as to meet with any conveyance, but that is very doubtful, and the disagreeable reflections this doubt occasions, contributes not a little to make me uneasy. However, should I be under the necessity of staying longer for want of an Opportunity of coming home, I can employ my time to advantage, without any additional expence, except that of my board, as I am a perpetual pupil to both Pott & John Hunter; & I shall be to Fordyce; so that for the first expence I may attend them as long as I please. But I please myself with the Hope, that notwithstanding the present Gloomy appearances I shall meet with no difficulty in returning to my friends, & when that happy time comes, give them no cause to regret, the kind part they have taken to enable me to become useful in my Station.

I have had the honour of being elected a Member of one or two of the Literary Societies. I do not mention this out of Vanity, by no means, but to acquaint my kind Uncle, with what I know will give him pleasure, that I am taken some notice of by men of Science. I am under great Obligations to Dr Fother-

gil; he continues his wonted kindness, as also do my other Friends.

In several of my late letters I acquainted thee particularly with my necessary expences, and that my finances would not be sufficient for them, and as thou wert kind enough to tell me thou would if there was real occasion, make an addition to them; I took the liberty to request a small one, which I hope thou wilt excuse, as I do not ask for it without an absolute necessity. My giving fifty Guineas to the Hospital, besides paying for all the different Lectures, Cloaths and board, has left but a very small sum, & I know not how much it may cost me to come back to America. A small sum now would be of great consequence to me, and if Providence spares me to return, I doubt not but what I should be able to repay it hereafter. There are several things that I have omitted for the sake of Oeconomy that I was very anxious to attend. A Course of Lectures was given on Natural history, which I never think of without regretting that it was not in my power to attend them; I may say the same with regard to those delivered on Natural Phylosophy, and the Lectures on Botany. These would have cost but a little, but that little was more than I could spare, being ditirmined to avoid all expences, that were not absolutely and indispensably necessary, to compleat me in my profession.

Surgeons are now much wanted here, for the Army & Navy, I have had very advantageous offers, but every consideration induced me not to hesitate a moment in rejecting them, tho' I am sorry to say some of my countrymen pursued a different course.

James Hutchinson to Israel Pemberton

London 6mo 12th 1776

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D^{r} Fothergill was in the Country on my Arrival in London, but was kind Enough by a few lines to advise me to enter S^{t} Bartholomew's Hospital, as a Dresser under Pott; but as the fee for this was fifty Guineas, I hesitated about doing it, and acquainted him with the state of my finances, and with the Opportunities I had of improvement in the Pennsylvania Hospital. He soon afterwards came to town, and we conversed on the Subject. He continuing to think that it would be best for me to enter as a Dresser, I could not therefore well avoid it, without acting in direct opposition to his Opinion, which I was by no means willing to do, knowing that his friendship would be of great service to me. He likewise proposed the making Surgery the object of my principal Attention, and to endeavour to qualify myself well in that particular branch, so as to become a good Operator, and if possible to render myself capable of giving a course of Lectures on that subject in Philadelphia, should there be a proper opening for the purpose, which we thought might probably occur. Therefore besides the Hospital, I immediately entered for two Courses of Anatomical Lectures and Dissections with D^{r} Hunter; for the first course of Lectures I paid four Guineas, for the second three, and for the Dissections five. Bodies to dissect during the winter cost between five & Six guineas more. Potts' Lectures on Surgery I attended, but being his pupil those were Gratis. I entered the Hospital in September, and the above Lectures in October. In November John Hunter (brother to D^{r} Hunter) began a Course of Lectures on Surgery & Comparative Anatomy, which D^{r} Fothergill though it best for me to hear; these were five guineas.

My time was employed on these subjects during the Winter; in the morning dissecting, dressing, the Hospital, and Potts Lectures took up my attention, and the afternoon the Anatomical course by D^{r} Hunter, and the Surgical one by his Brother found me employ, while the very little leisure that these avocations left, was spent mostly in copying my notes, and arranging those Ideas in proper Order, that were communicated to me.

Thus I passed the Winter and part of the Spring, lodging at the House of Benjamin Webb, a friend that lives in the Vicinage of Bartholomew's Hospital, to whom I was recommended by John Elliot and John Townsend. The meeting nearest my lodgings is the Peele, at which I delivered my Certificate.

In April and May all the above Lectures were finished, and I entered with Fordyce on the Materia Medica, Chemistry, & the Theory & Practice of Physic, for which I gave nine Guineas, and with Denman on Midwifery for which I paid seven more. These I now attend, and Fordyce & the Hospital take up the morning & Denman part of the Afternoon. I also hear Curtis's Lectures on Botany, a ticket for which was presented me, but his herborising excursions may probably come to about one Guinea & an half. These do not keep me so closely employed as I was in the Winter; however I have now but little time to spare. My Year at the Hospital will not be up till the middle of September, but the Lectures will be all concluded by the middle of August, when I mean to return if possible.

Thus have I been engaged, and I flatter myself to good purpose. have I had great Opportunities of improvement in Surgery, which I endeavoured not to neglect, and if providence Blesses those endeavours with success, I hope to be able thro' his Assistance to be of service to my fellow Creatures, and to lessen the sum of human Afflictions. However, whatever may be my Fate or Situation in Life, I shall always gratefully remember, that to my dear Uncle, next to my Creator, I am indebted for the means of improvement, and sincerely hope I may be enabled to conduct in such a manner, as to render myself in some degree deserving of the manifold favours I have received.

The concern Hutchinson expressed in these letters about getting home was real. It was not easy for a civilian to find passage from England to America in the summer of 1776. The packets no longer sailed; merchantmen risked capture. Some students, desperate to get home, enlisted as surgeons or surgeon's mates in the British Army, intending to desert as soon as they reached America. Hutchinson inquired into every sailing he heard of, his anxiety increasing as his expenses mounted—already greater than his uncle had expected. Then he heard there might be a chance of getting home from France. Fothergill gave his approval, and offered advice on professional and Friendly conduct:[7]

> There is no doubt of thy getting into immediate employment—but alas the Hospital—I wish it may not be in other hands—at least be filled with the wounded and distressed. This is one reason that makes me urgent for thy return. Most probably the Post of Surgeon to a Regiment may be offerd; if it can be decently declined it would be best, notwithstanding the present advantages. It would be out of character. In the Hospital thy services would be great, and thy progress tho more slow would be more certain—but all this must be left, for alas, we know not whether that great city may not be now in Ashes, or a heap of ruins!

Hutchinson made his way to Nantes where he succeeded in getting passage on the *Sally,* bound for Philadelphia with military stores for the rebels, and where, just before she sailed, Dr. Benjamin Franklin arrived, resting a few days en route to Paris. Franklin gave Hutchinson dispatches to carry to Congress.[8]

As Fothergill recommended and doubtless Israel Pemberton helped effect, Hutchinson was appointed a physician to the Pennsylvania Hospital a few weeks after his return. At the end of 1777, however, he left the post to join the army as a surgeon, and thereafter held several medical posts under the state government. In 1779 he was reelected to the Hospital staff. In that capacity, as the managers wrote in their memorial, he "cheerfully devoted the last 14 years of his valuable time in performing successfully the most difficult & disagreeable duties of Surgery, by which he was made . . . the Instrument of saving many lives." [9] Though he practiced surgery, he never taught it. In the organization and reorganization of the Medical School in 1789 and 1791 he took the chair of chemistry, which was an interest that antedated surgery.

Hutchinson fell ill on August 30, 1793; he was treated first by Adam Kuhn, then by William Currie; but he rejected the prescriptions of Dr. Rush. He died on September 6. The death of so prominent a person, and a doctor at that, made a profound impression on the public, while his professional colleagues long debated whether he had died despite, or because of, the treatment he received.[10]

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[7] Fothergill to Hutchinson, September 9, 1776.

[8] Hutchinson to Franklin, "Thursday," Franklin Mss., XL, 165, American Philosophical Society. A fragment of Hutchinson's journal, kept on his return voyage, February 25–March 16, 1777, is in American Philosophical Society.

[9] Pennsylvania Hospital Managers, Minutes, November 25, 1793.

[10] John H. Powell, *Bring Out Your Dead* (Philadelphia, 1949), *passim.*

A Medical History of Poison Ivy (Rhus Toxicodendron)[1]

By JEANNETTE R. MUNSON, B.S.[2]

POISON ivy, *Rhus toxicodendron,* the biggest plant nuisance in this country, is a native North American plant. The early European settlers found it where they landed on our shores and upon touching it were unpleasantly surprised several hours later as they became the first white men to experience poison ivy dermatitis.

"The poisonous weed, being in shape but little different from our English ivie; but being touched causeth reddness, itchings and lastly blysters, the which howsoever, after a while they pass away of themselves without further harm; yet for the time they are somewhat painful, and in aspect dangerous, it has gotten itselfe an ill name, although questionless of noe very ill nature." (1) (Capt. John Smith, 1609)

We can only assume that Captain Smith was not a sufferer—were he, he would not have questioned the "ill nature" or blamelessness of the plant!

This plant, a member of the family *Anacardiaceae,* is widely known for its dermatogenic properties. Some of the less offensive family members, such as the cashew and pistachio nut are great taste delicacies. The India ink tree, Hawaiian mango, Japanese lac tree, poison oak and poison sumac are also dermatogenic, but to a much smaller degree, and are also members of this family (1).

Since poison ivy sensitivity is so wide spread, and since for many years the reasons for its dermatogenic principles were unknown, American medicine has been much involved with the plant in many ways throughout its history.

The poison ivy plant was known to the American Indians both as a poison and as a medicinal agent (2), and Thompson (3) has shown that the North American Indian and Negro believed that the plant gives off invisible colorless vapors—which, when breathed or when they touch the skin lead to dermatitis.

The plant was first described, botanically, by J. P. Cornut (1) in his book *Canadensium plantarum, aliarúmque nondum editarum historia,* published in Paris, France, in 1635. This author was never actually in Canada, and based his work on plant collections made by Cartier and Champlain. Apparently neither of these men was poison ivy sensitive since no mention is made of the toxic properties of the plant! Cornut mistakenly believed that the plant was related to the English ivy and hence the nomenclature.

No further mention of the plant was made in the seventeenth century, but with the dawn of the eighteenth century, with the growing importance of American medicine and the rising numbers of written medical records and journals, accounts of the poisonous properties of the plant began to appear. In 1720, Dudley (4) wrote:

"....the Juice stinks as bad as Carrion ... it poysons in two Ways; either by touching or handling or by the Smell ... (but) it has effect on only some particular Persons and Constitutions. This sort of Poyson is never mortal, and will go off in a few days of itself."

In the same year, Drs. Sherad and Dudley (3) reported to the Royal Society of London several cases of Rhus poisoning caused by the smoke of burning wood. They did not

[1] Edward Bell Krumbhaar Prize Essay VII, Section on Medical History, The College of Physicians of Philadelphia, 1966.

[2] Class of 1967, Temple University School of Medicine, Philadelphia, Pennsylvania 19140.

mention that the plant had been burned, but seemed to hold the wood and the vapors responsible for the poisoning. There is, however, even today, considerable confusion of the botanical names of the poison ivy plant and that of the poison sumac and oak. Therefore, it is conceivable that the burning wood was indeed poison oak or sumac and that poison ivy was not involved at all.

Toward the end of this century, the journals being filled with reports of cases, researchers set out to find out what and why the plant caused dermatitis. Von Adelung (6) believed that the poisonous principle of the plant was a hydrocarbon which was 'extruded' at night or on cloudy days only. He reached this conclusion by experimenting with his brother who was hypersensitive. When the hypersensitive hands were placed inside an ordinary bell jar containing the plant—but not touching the plant—no dermatitis occurred. When the bell jar was covered with a dark cloth, and the hands were again placed inside the jar, dermatitis occurred. Thus, dark and mysterious vapors!

Horshfield (7) wrote a "definitive" work on all aspects of poison ivy in 1798 in his thesis submitted for the degree of Doctor of Medicine. He begins with a description of *Rhus toxicodendron*, and speaks of susceptibility of certain persons:

"...Irritable Habits are more liable to be poisoned than others, yet even among persons whose irritability is nearly alike, the tendency to be acted upon by this poison is very various..."

He also recognizes the immunity of animals who eat the plant. The author felt that females were more prone to develop the dermatitis during or immediately after a full meal. Horshfield believed that the juice of the plant was exhaled in the form of a gaseous fluid possessing its specific poisonous qualities, and thus surrounding the plant by an "atmosphere of poison." An acquaintance of Horshfield's,

FIG. 1. The first known illustration of poison ivy —from Cornut's book on the flora of Canada (1635). Copy in the Library of The College of Physicians of Philadelphia.

Kalm (7) tells the former of a patient he knew who merely by inhaling "the poisonous exudate of the plant ... was swelled to such a degree that he was as stiff as a a log of wood and could only be turned about in sheets."

The aspiring physician believed that concomitant with every case of poison ivy was a "sympathetic fever of the arterial system" and he speaks of the periodical return of most, sometimes all, of the symptoms of the disease about a year after its first appearance without fresh exposures. He seems to feel that this is in some way connected with the arterial fever and states that this may repeat itself for as many as ten years—thus, he called poison ivy, "an annual fever of the cutaneous system."

Medical treatment in these years was dominated by the figure of Dr. Benjamin Rush, as indeed was all of American medicine. As we might suspect, therefore, in the case of a violent case of ivy poisoning, Dr. Horshfield dutifully recommends bloodletting. This was said to shorten the course of the disease and render the symptoms less distressing. The poor patient was to drink large amounts of sea water to promote "copious and continued evacuation from his bowels" and he was to be exposed to plenty of "cold" in the form of air, water, or ice, since this was thought be "palliative of all symptoms". One wonders why a person with poison ivy would want to be further victimized!

With the beginning of the nineteenth century came many works and much writing on poison ivy but little increase in knowledge about the dermatitis. Many theories about the poisonous nature of the plant were advanced but one was no better nor more realistic than the next. Lavin (6), improving on von Mons, said that the night exhalations were not a hydrocarbon. However, the poisonous aura about the plant was still very much there:

"There is an atmosphere generated around these plants, which by simple inhalation excites its peculiar inflammation in the system. The gas, when not operated upon by the rays of the sun, posesses the power of causing vesicular eruptions when inhaled. Some peculiar temperaments are not liable to its effect. In the very commencement of the disease, it will be most favorable to premise a full bleeding." (8)

Rhittell (6) called the poisonous principle "rhustannic acid" and described it as a volatile toxic alkaloid. Maish (6) coined the term "toxicodendronic acid", a name that was to be in wide usage for many years in nomenclature of the poison. Park (2) further said that persons who had;

"...dark complexion, dark hair and spartic habit—(some) enjoy perfect immunity...light complexioned, blue eyed, light haired, and those of a phlegmatic temperament are perhaps more liable than others...(to poisoning by) toxicodendronic acid of the plant."

Burrel (3), apparently a non-believer, threw aside the chemical theories and found *bacteria* in the sap of the poison ivy plant:

"A lady was severly poisoned with Rhus toxicodendron. The exudating serum fom the inflamed part, and the leucocytes from the miliary vesicles were found to contain swarms of bacteria of the genus *Micrococci* as defined by Cohn. It was demonstrated that these organisms transferred from the inflamed to the healthy parts of the body communicated the severe malady, that they rapidly increased in number and were a true *contagium vivum*....similar organisms were found in the washings of fresh leaves and in the juice of the poisonous plants....The organism is spherical, usually single, but often seen in twos, seldom in strings of four or more about one half micron in diameter."

Pfaff (5) was the first worker to come close to the modern concept of the poisonous principle when he described a nonvolatile oil in the leaves, branches and roots which caused typical Rhus poisoning when applied to the skin. He tells of a patient who, in an effort to treat his case of poison ivy, rubbed his lesions with sweet oil. Later, when the patient used the oil to treat another ailment—windburn of the face—he developed a dermatitis from the oil exactly identical to the Rhus dermatitis. Pfaff concluded that the poisonous principle was soluble in the oil. Unfortunately Pfaff cried in the wilderness for many years, and older beliefs were still harkened to. Smith, in his books on poisonous plants, called the poisonous principle "pyrogallic acid" in 1905 (9) and "toxicodendrol" in 1923 (10). Frost (3), as late as 1913 was still finding bacilli in all the plant leaves. But he had support for his theory, too—"incubation period, complete natural immunity, hypersensitivity in the face of decreased natural resistance, febrile symptoms, and anaphylaxis."

Persons afflicted with sensitivity to the disease were among the most unfortunate of the era, for as we have seen the treatments prescribed were legion and rather strenuous. However, it is suprising to note that as early as 1829, Dakins described the first mentions of prophylaxis, which was and still is being experimented with (11):

"Some good meaning, mystical, marvellous physicians or favored ladies with knowledge inherent say the bane will prove the best answer, and hence advise the forbidden leaves to be eaten, both as a preventative and cure to the external disease."

Dakin's scepticism was well taken, for many cases of oral dermatitis were reported (3). Dakin besides was a sound country doctor who believed that the "poison," as the country folk called it, was an ailment to be borne: "a thing not dangerous, though troublesome."

Treatment centered around resolving the local disturbance, but precautions were taken to rid the body of the poison, since as late as 1913 some persons believed that the toxic principle became blood-borne and was thus disseminated (6). Each patient got some sort of sedative for pain, he was relieved of some blood, and he got a thorough purging, "for the purpose of removing any offending matter from the stomach and bowels and increasing the action of secremints and absorbants (3)." Topical remedies were legion, then as now; they ranged from mud to exotic secret preparations, the ingredients of which were known only to their quack concocters. One thing probably did as much good as the next; if they relieved the itching and other symptomatology, they were worthwhile. At least they made the patient feel better, which is more than can be said for the internal remedies.

In 1913, Guernesy (12) said that he did not believe that the poison entered the circulation; he believed it to be a local affectation only. He described the sensation of "thousands of fleas traveling and biting at the same time" as the cardinal feeling of the patient! He recommended wrapping the patient in his entirety in thick folds of gauze impregnated with 50% alcohol and powdered sugar of lead. The patient was then not to move. One wonders if he could!

Medicine's strangest tales of *Rhus toxicodendron* lie in the annals of pharmacology. It all began in 1788 when Du Fresney (13), a French physician and lecturer in botany, introduced *Rhus toxicodendron* as a medicine. He tells the story of a young American who, upon recovery from a case of Rhus dermatitis, found himself cured of chronic eczema of six years standing. Du Fresney, an emigrant to America, experimented further and found that pemphigus, eczema and other "obstinate" skin diseases benefited by a case of poison ivy! One of his colleagues, a Dr. Phillips (13) found that *Rhus toxicodendron* poisoning was a wonderful remedy in erythema and erysipelas, especially when they were accompanied by vesicles and bullae.

Homeopathic medicine was prominent at this time and had assumed leadership of one main stream of American medicine. Those physicians who followed the dictums of this school of thought felt that Rhus poisoning would be very beneficial since the signs and symptoms elicted by an attack of Rhus poisoning were very similar to the signs seen in other skin diseases: swelling, edema, redness, etc. Since Nature's mechanisms were imitated, creation of this natural defense reaction by the use of *Rhus toxicodendron* should be valid in curing other diseases. Therefore, Rhus tox, as it was called, became important pharmacologically as an internal as well as an external medication.

Most physicians felt that taken internally, the Rhus tox acted as a diuretic. Horshfield (7) describes the case of a woman who had "universal anasarca", and who received Rhus tox, intravenously, as an emergency measure. "The infusion

produced copious perspiration."—and apparently cured the patient. He prescribed the Rhus tox for phthisis pulmonalis, mania, melancholia and epilepsy. Also, he used Rhus tox to cure herpes and other skin conditions, upper extremity palsies, palsies following apoplexy and convulsions. Horshfield had a special feeling for the drug, no doubt, since the weed had helped to earn him his degree; nonetheless, such devotion is almost unbelievable.

Many recipes were available for preparation of the medicinal extract and in 1879, it was on the secondary list of the United States Pharmacopeia (2). A tincture was said to be in official use by the Germans (2). Gibson (14) tells the story of many persons who were "in the last stage of consumption" and who were restored to perfect health by taking the "decoction of a vine... the fact (being) too well established to admit of any doubt." The vine—of course—*Rhus toxicodendron.* Burgess (13) lists the therapeutic actions of the Rhus tox as:

"tetanic, stimulant, narcotic, diuretic, diaphoretic, laxative, alternative, and should be used mainly for rheumatic afflictions of the fibrous type."

In these rheumatic afflictions, it is to be used both internally and externally. He also states:

"In all cases of paralysis dependent on a torpid condition of the nerves, it is said to have produced beneficial results—the first symptoms being prickling and twitching in the paralyzed part."

Strange that it should prickle—but then, the patient had poison ivy, and would have an itchy arm!

In 1885, Rhus tox was first on the list of remedies for puerperal fever. Aulden (16) gives us an account of the action of the drug:

"After the exhibition of the drug for a few days, the urinary flow is decidedly increased and the acid character is modified to that extent that no disagreeable symptoms attend upon micturition; the appetite improves, while she who has suffered torments in trying to secure much needed rest is lulled into a repose which is not unlike that of early childhood."

Aulden (17) also says that the major effect of the drug is on the cells of the intestinal tract and gives regulations for the gathering of the leaves for the preparation of the drug:

"(the drug) should be made from fresh leaves... gathered during periods of efflorescense from among plants grown in shade or before the sun strikes (the) plants... (since) the active principle, toxicodendronic acid, is an extremely volatile substance."

He claims to have found the drug to be useful in "allay(ing) the irritability of the sensory nerves", and he lists twenty four other maladies, including relief and disappearance of hemorrhoids which are allayed by its use.

Royal (15) speaks highly of Rhus tox, citing its power to prevent disease even when all conditions are favorable for its development, its ability to combat disease after it has involved the system, and sees the "homeopathicity of the drug to septicemia when given internally."

In the 1900's, Rhus tox became "a veritable sheet anchor in ophthalmological work!" Deady (18) claims the value of the drug in almost any eye condition. The homeopathicity of its effect was considered to be its major value:

"After the operation for cataract, one of the first symptoms indicating danger is oedematous swelling of the lids and no drug in the materia medica compares with Rhus for insuring the safety of the eye. When the pathological process becomes advanced, even to the appearance of pus within the eyeball, still we may confidently rely on this remedy which has cured many such cases when they were apparently hopeless. In all postoperative complications it is of greatest value and too much emphasis cannot be placed upon this statement. When a second eye takes on a sympathetic irritation from its diseased fellow, it is first manifested by... swelling... (and) more or less profuse lachrymation, the latter another valuable invocation for Rhus tox... it is the first drug to be thought of in that extremely dangerous situation!"

He goes on to list at least twenty other acute eye conditions for which Rhus tox is the drug of first choice and best results. Rhus tox even will cure "paralysis of certain ocular muscles, especially if due to wet or damp," according to Aulden.

The Rhus tox was also used to cure the dermatitis, and many an unusual story is told of this affliction. In 1838, for example, a "certain young lady" got a case of ivy poison and did so again every year thereafter until 1851 when she got a recurrence (20). These recurrences were supposed to occur without fresh exposure and were thought to be less severe but more difficult to cure and more prone to chronicity (13).

White (21) wondered about the course of the disease and if it was a merely cutaneous one. He told of a case of "congenital" poison ivy in a child born to a mother who had dermatitis. The child a few weeks later developed a rash on his abdominal skin, exactly like that on his mother's arms. Dr. White did not believe that the disease was communicated from the mother's arms and body to the child. He cited the fair complexions of the mother and father and seemed to feel that this would lead to the dermatitis. He concluded: "Is it not probable that with the mother it (Rhus dermatitis) became constitutional and was then communicated to the child in utero and developed in it afterward?" Now we know that the child must have been contaminated by the mother but, in 1886, one doctor could only go by what he could see, and considering that a lot of other people were "seeing" emanations from plants, auras of poison, and microbes in the plant sap of the Rhus plant, he was seeing a bit more clearly than some of his colleagues.

The nineteenth century brought little increase in the knowledge about the dermatitis, but with the dawn of the twentieth century, ideas and concepts regarding allergic diseases were being formulated and it was realized that poison ivy dermatitis is an example of this type of mechanism. The active dermatitis-producing principles of Rhus toxicodendron are a group of closely related catechol derivatives. When placed on the skin, they act as haptenes and are part of an enzymatic adaptation which occurs in the epidermal cells. After the antigen forms, it is passed on to descendents of the cells. Physicians and patients alike know a great deal about the mechanism—but they still can't cure it!

"We struggled toughly upward, canted to and from by the roughness of the trail and continually switched across the face by sprays of leaf or blossom. The last is of no consequence at home; but here in California it is a matter of some moment. For in all woods and by every wayside there prospers *an abominable shrub,* or *weed,* called Poison...."

Robert Louis Stevenson, 1883.
Tramp in a California Wood (3)

REFERENCES

1. Rostenberg, Adolph: An anecdotal biographical history of poison ivy. Arch. Derm. **72**: 438–445, 1955.
2. Park, Roswell: Dermatitis venenata; or Rhus toxicodendron and its action. Arch. Derm. **5**: 227–234, 1879.
3. McNair, J. B.: *Rhus Dermatitis,* U. of Chicago Press, Chicago, 1923.
4. Dudley, Paul: An account of the poyson wood tree in New England. Philosophical Trans. (London) **31**: 125, 1720–1721.
5. Scott, J. Allison: A case of poisoning acquired in an unusual manner. Phila. Med. J. **3**: 902–904, 1899.
6. von Adelung, E.: An experimental study of poison oak. Arch. Int. Med. **11**: 148–164, 1913.
7. Horshfield, J.: Rhus vernix, Rhus radicans, etc., Philadelphia Medical Theses, 1798.
8. Christy, Asa: An essay on the poisonous qualities of some species of the genus rhus. N. Y. Med. & Phys. J. **8**: 674–723, 1829.
9. Smith, A. B.: *Poisonous Plants of all countries.* John Wright & Co., Bristol, 1911, p. 47.
10. *Ibid:* p. 66.
11. Dakin, R.: Remarks on a cutaneous affection produced by certain vegetables. Am. J. Med. Sci. **4**: 225–6, 1829.
12. Guernesy, J. C.: Rhus poisoning. Hahnemannian Monthly **48**: 161–174, 1913.
13. Burgess, T. J. W.: Poisonous plants of Canada. Canad. J. Med. Sci. **5**: 327–334, 1880.

14. Gibson, George: Observations on the internal use of the Rhus radicans. Phila. Med. & Phys. J. **6**: 478, 1804.
15. Royal, George: Use of Rhus tox. Med. Cent. **3**: 52–53, 1895.
16. Aulden, J.: Therapeutic Use of Rhus toxicodendron. Therapeutic Gaz. **5**: 679–682, 1889.
17. Aulden, J.: The use of Rhus toxicodendron. Med. & Surg. Reporter **63**: 2–3, 1890.
18. Deady, C.: Pathogenic and therapeutic action of Rhus tox upon the eye. J. Ophthal., Otl., Laryngol. **12**: 352, 1900.
19. French, J. M.: A case of Rhus poisoning. Chicago Medical Times **35**: 512–515, 1902.
20. *Ibid:* A case of annual return of cutaneous poison. N. J. Medical Reporter **55**: 326, 1851.
21. White, G.: Is Rhus poisoning a purely local affection? Med. Record **30**: 689, 1886.

Memoir of George William Norris (1875–1965)*

By T. GRIER MILLER, M.D.

GEORGE WILLIAM NORRIS was a charming, modest, aristocratic gentleman. His interests varied from time to time, but fundamentally all were based on his love of nature. He had many hobbies, medicine being only one of them. Even in his chosen profession he was more interested in the person who had the disease and in the techniques for studying him than in the disease itself. He had an equal fascination for landscapes, forests, streams, flowers, the cultivation of the soil and wildlife.[1] All of these interests are manifest in the results of another of his hobbies, photography, one that was initiated and encouraged by his father.

Dr. Norris' choice of medicine as a profession doubtless was influenced by his family background. The first member of his American family was Isaac N. Norris, a Quaker, who immigrated to Philadelphia, by way of Jamaica, in 1693. Isaac's son, Charles, was one of the original Managers of the Pennsylvania Hospital (1751) and its second Treasurer (1752–56). George's grandfather, George Washington Norris, was a surgeon to the same hospital and Professor of Clinical Surgery at the University of Pennsylvania, author of "An Early History of Medicine in Philadelphia" and a founder of the Pennsylvania Historical Society. His father, William Fisher Norris, was a surgeon in the Civil War, had training in ophthalmology in Austria and became the first Professor of Ophthalmology at the University of Pennsylvania.

Dr. Norris was born in Philadelphia on January 1, 1875, but his mother, Rosa Clara Buchmann, was Viennese. He spent his summers during early childhood in Vienna, where he learned the German along with the English language. He attended the George F. Martin's School in Philadelphia, where, as he later recalled, he was taught "to concentrate, to study and to memorize". He had training in Latin for six years and in Greek for four. In College at the University of Pennsylvania, in spite of his good preliminary education, his only interest was in physics and he has stated that he studied other courses only enough to pass. Indeed he once admitted his bewilderment about mathematics to his professor and was told "Do your best, Mr. Norris. I will make allowances, remembering your father". In the University of Pennsylvania Medical School, however, he worked hard, even to the detriment of his health, and following graduation in 1899 spent two years as a Resident at the Pennsylvania Hospital. During that time he published two papers: one on catalepsy and one on cocaine poisoning. Subsequently he became associated with the dispensaries of the Episcopal, the Orthopedic, the Polyclinic, the Pennsylvania, the Philadelphia General and the University Hospitals; also with the Phipps Institute. Then, as an Instructor and later as an Associate in the University Medical School, he began his teaching, especially in physical diagnosis. In 1912 he was made an Attending Physician to the Pennsylvania Hospital and subsequently did most of his teaching at the bedside in that institution. Meantime he had pub-

* Read before The College of Physicians of Philadelphia, 6 April 1966. Prepared and published at the request of the Council of The College of Physicans of Philadelphia.

[1] Personal diaries are available in the libraries of The College of Physicians of Philadelphia, the Pennsylvania Hospital, and the University of Pennsylvania.

lished 23 papers, mostly on cardiac and pulmonary diseases. To some extent these resulted from exhaustive study of autopsy records in the various hospitals and the available literature, but mostly were based on meticulous physical examinations. An outstanding publication for that era was one of the first on sphygmographic tracings in patients with heart disease.[2]

In spite of increased teaching and the development of considerable practice he published during the next few years three books: "Studies in Cardiac Pathology" (1911); "Blood Pressure, Its Clinical Applications" (1914, 4 editions) and, with H. R. M. Landis, "Diseases of the Chest and the Principles of Physical Diagnosis" (1917, 6 editions).

Meantime, in 1911, Dr. Norris joined the Medical Reserve Corps of the Army and in 1916 had some training at Plattsburg. In April of the following year, as a Captain and Chief of the Medical Service of the Pennsylvania Base Hospital Unit No. 10, he went to England. This organization two months later took over the British Hospital No. 16 in Le Tréport, France. There, as a Major, he functioned for ten months. Then he was transferred to the AEF and made a Lt. Colonel and Senior Medical Consultant for the Divisions in the Toul Sector; later, as Consultant to the Fourth Army Corps, taking part in the St. Mihiel Offensive. He became a full Colonel in November of 1918 and was Commander of Troops on the S. S. Saxonia, the first hospital ship to return to America after the war. He was honorably discharged from the Army on December 27th, 1918, and in March, 1920, received a citation from General Pershing for "exceptionally meritorious and conspicuous services".

Following the war Dr. Norris resumed practice and teaching. By 1922 he had contributed a total of 50 articles to medical journals. By that time he had become Professor of Clinical Medicine at the University and a Chief of Medical Service at the Pennsylvania Hospital. He excelled as a teacher, always emphasizing the importance of a meticulous physical examination, of methods of learning rather than of accumulating knowledge; not of doing more but of doing better. He appreciated more than many of his time the importance of a scientific approach in medicine.

In 1931, due largely to impaired hearing, Dr. Norris gave up his teaching and consultations. Subsequently, except for the winter months, he lived on the beautiful country estate that he had inherited from his father. That was "Woodbourne", Dimock, Susquehanna County, Pennsylvania. There he pursued certain of the hobbies that had fascinated him from childhood. He no longer could do the bicycling, the sculling, the horseback riding, the figure skating, the cricket and the mountain climbing of his boyhood, but he reveled in the mountains, the valleys, the forests, the streams, the crops, the flowers, the animals and all the favorite activities of an intelligent and alert country gentleman. His interest in photography, which had been helpful in his teaching and in illustrations for his books, persisted. Also during this period he accumulated, classified and bound together many beautiful pictures that he had taken during his earlier life: of family and friends, of current costumes and homes, of Alpine and other foreign tours.

Dr. Norris maintained throughout life a deep interest in many of the organizations to which he had been elected: medical, social and business. In addition to his membership in the Association of American Physicians (1912–), The College of Physicians of Philadelphia (1905–), The American Clinical and Climatological Association, the Interurban Clinical Club, the American Tuberculosis Society (a founder member), the Philadelphia Heart Association (a founder member), the Na-

[2] University of Pennsylvania Medical Bulletin 23: 425, 1910–11.

tional Association for the Prevention of Tuberculosis and some other medical organizations, he belonged to the Philadelphia Club, the Racquet and the Barge Clubs, the American Philosophical Society, the Academy of Natural Sciences, the Society for the Advancement of Agriculture (1912–), the National Wildlife Federation and the Historical Society of Pennsylvania. Also for 55 years (1908–63) he was a trustee of the Mutual Assurance Company, as had been five of his forebears.

Even when living in Dimock, Dr. Norris made routine visits to Philadelphia for special meetings of certain of these organizations. Not infrequently at such meetings he made addresses, usually of a historical nature, and sometimes in verse. Once he presented a humorous poem at a party in honor of a poor German who had come to this country as a stowaway on a ship, but whose name now identifies a permanent Committee in the University of Pennsylvania Hospital for gifts to patients—the Abrahamson Committee. Also he gave a five page address in verse at an internes' celebration in honor of the 200th anniversary of the Pennsylvania Hospital. Another fascinating address was made at a meeting of the Colonial Dames of America, in which he presented pictures, with full descriptions, of all the trees, shrubs and flowers that were native to his section of northern Pennsylvania.

Dr. Norris died on December 11th, 1965. He is survived by his wife, Annie Bradford Hays, whom he married in 1956. Most of his contemporaries have predeceased him, but their admiration and affection for him are manifest in many recorded statements. Those who were his internes and younger associates will never forget his generous, helpful and friendly attitude toward them. For those of us who knew him less well he will be remembered for his retiring nature, his kindness, his sense of humor and his dedication to a better life for all mankind.

Memoir of A. Bruce Gill (1876–1965)*

By GEORGE M. LAWS, M.D.

ARTHUR BRUCE GILL was born of Scottish ancestry on December 12th, 1876, in Greensburg, Pa. He graduated from Muskingum College with a B.A. degree in 1896 and from the University of Pennsylvania with an M.D. degree in 1905. He was a member of the Sigma Xi and Alpha Omega Alpha honorary fraternities. Dr. Gill interned at the Presbyterian Hospital in Philadelphia and practiced here throughout his active professional life.

Dr. Gill's early hospital appointments were in general surgery, his later ones in orthopedics. He was Assistant Surgeon at the Presbyterian Hospital, 1906–1914, Orthopedic Surgeon, 1914–1941, and Consulting Orthopedic Surgeon, 1941–1953. At the Widener Memorial Industrial School for Crippled Children he was Assistant Surgeon, 1911–1920, and Chief Surgeon, 1920–1942. He was Orthopedic Surgeon at the Episcopal Hospital, 1915–1925. At the Orthopedic Hospital he was Assistant Surgeon, 1908–1914, and Orthopedic Surgeon, 1914–1941. He was Chief Surgeon at the Children's Seashore House, Atlantic City, N.J., 1938–1943, and at the Alfred I. DuPont Institute, Nemours Foundation, Wilmington, Del., where he was later a member of the Medical Advisory Board. He was also operating surgeon for pediatric clinics in Carbon and Schuylkill counties, 1927–1945. From 1920–1942 Dr. Gill was Professor of Orthopedic Surgery at the University of Pennsylvania School of Medicine; he was Emeritus Professor from 1942 until his death.

Dr. Gill was a member and officer of many organizations, including the American Academy of Orthopedic Surgeons and the American Orthopedic Association. He served as president of the former in 1938 and of the latter in 1944. He was also a member of the International Society of Orthopedic Surgery and Traumatology. He was a member and president of the Philadelphia Orthopedic Club and a Fellow of the Academy of Surgery of Philadelphia. He was elected a Fellow of the College of Physicians of Philadelphia in 1920. He held honorary memberships in the Ambroise Paré Society of France, the Pennsylvania Orthopedic Society, and the Orange County, Florida, Orthopedic Society.

Dr. Gill's special interest in crippled children led to his serving on various advisory committees concerned with their care, including those of the A.M.A., the Secretary of Health of Pennsylvania, the Children's Bureau of the Department of Labor, Washington, D.C., and the Department of Health of New York City.

In addition to his many contributions to the literature of orthopedic surgery, Dr. Gill developed a number of original surgical procedures, six of which bear his name. He was rated among the foremost authorities on congenital dislocation of the hip.

A tall, lithe, and athletic man, Dr. Gill took pride in having coached a winning football team at Muskingum. He liked tennis, swimming, and dancing, and he was one of the earliest members of the Doctors' Golf Association of Philadelphia, which he served as president in 1950. He was active in organizing the Inter-Hospital Bridge League. His other memberships included the Merion Cricket and Golf clubs, the Franklin Inn, and the St. Andrews So-

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ciety. He enjoyed playing classical music on the piano.

In 1938 Dr. Gill married Mabel Halsey Woodrow, a talented artist. He retired from practice in 1953, after which he was the victim of heart disease, cancer, and a stroke. Since 1955 Dr. Gill had lived in Mount Dora, Florida, under Mrs. Gill's devoted care. He died of congestive heart failure on November 7th, 1965, near Asheville, N.C., where he had spent the last few summers.

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Reiter's Disease: Its Nature and Relationship to Other Diseases[1]

By CURRIER McEWEN, M.D.[2]

MR. PRESIDENT, Miss Mildred and Miss Marion Bach, other relatives and friends of Dr. Bach, Ladies and Gentlemen:

I know you will permit me to begin my talk this evening by saying how much I appreciate the honor and the opportunity you have given me to be the first Theodore Franklin Bach lecturer. I met Dr. Bach at various meetings of the American Rheumatism Association but regret that I never had a chance to know him really well. The warm tribute to him written by Dr. Clyde Kelchner for the *Transactions and Studies* of this College has eloquently spoken of his many gifts and has added to my sense of responsibility this evening. During the all too rare conversations I had with Dr. Bach, the subject of Reiter's Disease did not come up. I am sure, however, that his inquiring mind must have been stimulated by the many questions which have surrounded that disease down through the years, some of which I shall touch on this evening.

The purpose of this discussion of Reiter's Disease is not to give a comprehensive presentation of the subject but, rather, to consider four aspects of it which are either controversial or of particular current interest, namely: 1. its history; 2. its terminology and the specificity of various manifestations; 3. its possible relationship to other diseases; and 4. its cause.

History

The combination of manifestations which is commonly referred to as Reiter's Syndrome or Reiter's Disease derives that name, of course, from the well known case report by Hans Reiter (1) of a young German officer in World War I who developed severe diarrhea and then, in rapid succession, urethritis and cystitis, conjunctivitis and iritis, arthritis and, probably, balanitis. Reiter correctly recognized the disease as distinct from other forms of arthritis. Six days prior to Reiter's published paper Fiessinger and Leroy made a verbal report before the Société Médicale des Hôpitaux de Paris of their comprehensive studies of an outbreak of bacillary dysentery among French troops on the Somme front. Included in their report (2) was a detailed account of what they referred to as the conjunctival-urethral-synovial syndrome

[1] Theodore F. Bach Memorial Lecture I, The College of Physicians of Philadelphia, 4 May 1966.

[2] Professor, School of Medicine, New York University, New York, New York.

which occurred in four of their patients with bacillary dysentery, and which, obviously, was the same entity as that described by Reiter. For some time there was speculation as to whether a new disease had appeared on the medical horizon in some way related to the war, but subsequently earlier reports of what, in retrospect, was the same disease came to light. Brodie (3) included summaries of five undoubted cases in his book on "Diseases of the Joints" published in 1818 and commented that, to his knowledge, they were examples of a disease which had "not been described by any pathological or surgical writer" previously. Moreover, most, if not all, cases of arthritis following bacillary dysentery appear to be the same entity. Manson-Bahr (4) attributes knowledge of such cases to Caelius Aurelianus in the 5th Century A.D. According to Huette (5), Zimmermann and Lepecq de la Clôture wrote of post-dysenteric arthritis in 1765, and Stoll in 1776 specifically mentioned dysuria as a feature of it. Thus, the question of the mysterious appearance of a new disease entity was set at rest. Following Reiter's article, interest in the disease grew slowly. The first report in the United States was that of Bauer and Engleman in 1942 (6). During the twenty-six years between Reiter's paper in 1916 and 1942, only 80 publications appeared and about one-third of those were primarily concerned with dysentery. In more recent years, however, interest has increased greatly and the twenty-four years since 1942 have witnessed approximately 900 reports.

In closing this brief summary of historical aspects of Reiter's Disease it should be noted that the long delay in its recognition as a distinct entity is not surprising when one recalls how recent it is that even the more common rheumatic diseases were clearly differentiated from one another. There can be little wonder that earlier cases were accepted as part of dysentery or as examples of gonococcal or rheumatoid arthritis. Indeed there are still some who consider it a form of the latter.

Terminology

Reiter's Disease has been given many names by various authors. In addition to the eponyms involving the names of Reiter, Fiessinger and Leroy, and Brodie, there are various descriptive terms. Reiter himself first called it "Spirochaetosis arthritica" after a spirochaete which he erroneously thought the cause. Other names include the conjunctival-urethral-synovial syndrome, arthritis urethritica, non-gonococcal urethritis with conjunctivitis and arthritis, post-venereal arthritis, subacute infectious polyarthritis and mucositis, infectious uro-arthritis and a number of other names related to dysentery. None of these is entirely satisfactory, and although Brodie clearly described the disease almost a hundred years before Reiter and, like the latter, recognized it as a distinct entity, it was Reiter's report that called it to modern attention and his name is so firmly attached to it that, until newer knowledge makes possible a suitable term based on its cause, it seems best to continue to call it Reiter's (or Brodie-Reiter-Fiessinger and Leroy) disease.

There is much disagreement as to whether the entity should be designated a syndrome or a disease. Those who prefer the former do so because of the possibility that it may have more than one cause. On the other hand, there are few entities in medicine with more distinctive features, and it can be reasonably contended that if any symptom complex of unknown cause deserves to be considered a disease this one does. A practical disadvantage of the term "Reiter's Syndrome" is the lack of agreement as to what constitutes the syndrome. Some authors (6, 7) insist on the "classical triad" of urethritis, conjunctivitis and arthritis, yet Reiter's original case had, in addition to that triad, diarrhea and balanitis; and other investigators with

TABLE I

Combinations of Manifestations in 71 Patients with Reiter's Disease

Non-Articular Features	Articular Involvement					
	Spondylitis	Sacroiliitis only	Peripheral joints only	Full "Triad"	No Urethritis	No Conjunctivitis
C . . .	4	2	.	14	3	13
C U . .	6	1	7	10	1	2
C U K .	5	1	4	6	7	3
C U K B	2	4	.	3		2
C U . B	1	2	.			1
C . K .	2	1	.			
C . K B	.	.	1			
C . . B	5	2	.			
. U . .	3	3	7			
. U K B	.	2	.			
. U . B	2	.	1			
. . K B	1	1	.			
. . . B	1	.	.			
Total Patients	32	19	20	33	11	21

C—conjunctivitis; U—urethritis; K—keratoderma blennorrhagicum: B—balanitis.

special interest in the disease (8, 9) are prepared to make the diagnosis if any reasonable combination of these seven features is present. The combinations presented by 71 patients considered by us to have Reiter's disease are shown in Table I. As noted in the table, 14 of these patients had the so-called triad alone and another 19 had the triad plus various other features, a total of 33. However, 38 of the patients lacked the full triad in the attacks tabulated. We believe that such a combination as urethritis, arthritis and balanitis or keratoderma is no less diagnostically acceptable than urethritis, arthritis and conjunctivitis and such combinations were present in 19 more of these patients. On the other hand non-gonococcal urethritis alone or conjunctivitis alone with appropriate arthritis would warrant merely a presumptive diagnosis of Reiter's Disease; and the 19 such cases included in Table I were accepted as definite examples of Reiter's Disease only because other features were present in earlier or subsequent attacks.

One other controversial diagnostic question is concerned with the presence of gonococci in the urethral pus. There are some who state that the diagnosis of Reiter's Disease must be excluded in such cases (11). This attitude appears to me to be no more reasonable, provided the features of Reiter's disease are distinct, than the exclusion of an obvious diagnosis of primary syphilis or chancroid because of concurrent gonococcal infection. Yet the same authors accept gonococcal infections as not incompatible with a diagnosis of rheumatoid arthritis (11, 12). Actually, I suspect that most examples of what they have called post-gonorrheal rheumatoid arthritis (12) are cases of concurrent gonococcal urethritis and Reiter's Disease. Certainly some attacks of rheumatoid arthritis can be expected to follow gonococcal infections if only through coincidence. We have noted gonorrhea only five times (0.7%) among 738 patients with rheumatoid arthritis in our files. In constrast, the files record 71 patients with Reiter's disease, of whom 12 (18%) had urethral cultures which were positive for gonococci—an experience which is not surprising in this

largely promiscuous group of young men. None of these patients gave positive tests for rheumatoid factors even with the very sensitive sheep erythrocyte agglutination-inhibition method (13).

Relationship to Other Diseases

a) Bacillary Dysentery: The long recognized occurrence, following attacks of bacillary dysentery, of a form of arthritis like that which we now relate to Reiter's Disease naturally led to the assumption that it was a complication of dysentery. This is considered more fully in the discussion of cause.

b) Gonococcal Arthritis: Because of the common occurrence of Reiter's disease following venereal infection, it was long confused with gonococcal arthritis; and it was not until after the report by Bauer and Engleman in 1942 that the distinction began to be appreciated in the United States. Prior to that time two types of gonococcal arthritis were recognized. One had the clinical appearance of a pyogenic infection of one or two joints, and, on aspiration, frankly purulent synovial fluid was obtained which frequently yielded gonococci on culture. In the other type a larger number of joints was involved in a more indolent process, and aspiration gave a comparatively clear fluid from which gonococci were never obtained. It was conjectured that the first type was caused by direct invasion of the joints by gonococci, whereas the second type resulted from an allergic reaction to some gonococcal product. Today those views are chiefly of historical interest although, as discussed in the next section, there still are some who invoke delayed sensitivity to gonococci and shigellas as the cause of Reiter's Disease.

Parenthetically one may mention here also the view that keratoderma blennorrhagicum is a manifestation of gonococcal infection. In all early papers on the subject that was the accepted cause. Today, in contrast, although there still are some who believe that keratoderma blennorrhagicum can be a manifestation of either gonorrhea or Reiter's Disease (14), the great majority now accept it solely as a feature of the latter. The same is true also of the "lacy" calcaneal spurs which were for so long thought to result from gonococcal infections.

c) Rheumatoid Arthritis: This has already been mentioned briefly in the comments on "post-gonorrheal rheumatoid arthritis". There are a few writers who contend that Reiter's Disease is merely a variant form of rheumatoid arthritis. If, however, one is to accept the latter as a disease characterized by reasonably distinct clinical features, there is little to support that view. Patients with Reiter's Disease do not have subcutaneous nodules, their joint involvement is often asymmetrical, and their sera give negative tests for rheumatoid factors (15), although synovial plasma cells may give positive results using the technic of immunofluorescence (16). Conversely, rheumatoid disease is not associated with diarrhea, urethritis, balanitis, or keratoderma blennorrhagicum, and eye involvement is rare in adults.

d) Ankylosing Spondylitis: The frequent involvement of sacroiliac joints in Reiter's Disease, progressing to spondylitis in some patients, has led a few writers to the belief that ankylosing spondylitis and Reiter's Disease are the same (17, 18). Our observations, however, do not support this view (19). Sacroiliitis is indeed a common finding in Reiter's Disease but even in cases with spondylitis it tends to advance very slowly compared with that of ankylosing spondylitis. The changes in the spine are still more distinctly different. In Reiter's Disease the typical bambooing of ankylosing spondylitis rarely occurs; instead, the syndesmophytes are scattered, are more often asymmetrical, are much thicker, and extend well beyond the margins of the verte-

bral bodies above and below. In these respects they resemble the syndesmophytes of psoriatic spondylitis (19).

e) Psoriatic Arthritis: The similarities between cases of Reiter's Disease with keratoderma blennorrhagicum and psoriatic arthritis may be very striking. In the first place, the lesions of pustular psoriasis and keratoderma often cannot be distinguished either clinically or histologically. As noted above, the spondylitic changes in psoriasis and Reiter's Disease are also similar. Most impressive of all are the occasional patients with perfectly typical Reiter's Disease and keratoderma blennorrhagicum which gradually converts to ordinary, plaque-like psoriasis (20–22).

Cause

a) Spirochaete: Reiter's early impression that the disease was caused by a spirochaete was soon abandoned by that author himself.

b) Shigella: The reasons for thinking that Reiter's Disease may be a manifestation of bacillary dysentery have already been mentioned. Reiter's own case and those of Fiessinger and Leroy clearly occurred in patients with shigella infections and there is little likelihood that these associations were purely fortuitous. The most extensive report of this association is that of Paronen (23) based on the epidemics of Flexner dysentery which occurred in Finland from 1943 to 1946. He collected data on 334 cases of Reiter's Disease which occurred among the roughly 150,000 persons with shigella infections (0.2%). It is not unnatural that he concluded that the cause of Reiter's disease is the shigella, as many others had before him. Indeed the possibility cannot be dismissed that the syndrome of Reiter's Disease can have several causes of which one is the shigella, and that the cases which are unassociated with diarrhea and appear to be of venereal origin are a different disease. It seems more probable, however, that both types of cases have a common cause and that the dysentery in some way enables this cause to become effective.

c) Secondary to Gonococcal Infection: It has already been mentioned that prior to the recognition of Reiter's Disease in this country in 1942, most American physicians considered it a special type of gonococcal arthritis caused by reaction of the joint to some product of the gonococcus rather than to pyogenic invasion and multiplication of the microorganism itself. This view that the disease is one of delayed hypersensitivity to the gonococcus remains one of the currently held theories (18). According to this concept the relationship of Reiter's disease to gonococcal arthritis would be analogous to that of rheumatic fever to hemolytic streptococcal pyogenic arthritis. This is an attractive hypothesis but it is purely conjectural, and serologic studies of patients with Reiter's Disease have failed to show evidence of gonococcal infection in any greater frequency than that of comparable groups of healthy promiscuous young men (24).

d) Mycoplasmas: Since the report of Dienes *et al.* (25) in 1948 there has been particular interest in the group of pleuropneumonia-like organisms (PPLO), or mycoplasmas, as the possible cause of nongonococcal urethritis and Reiter's Disease. In 1898 Nocord had first identified these agents as the cause of pleuropneumonia in cattle, and in 1937 Dienes and Edsall (26) isolated them from a Bartholin's abscess and thus demonstrated that they can cause disease in man. As information accumulated regarding various strains of mycoplasmas it became clear that they have a particular capacity to damage joints in a variety of animals and they became suspect as a possible cause of Reiter's Disease in man. They can be readily isolated from the genito-urinary tract of many patients with this disease in contrast to their ap-

parent absence from normal males. However, they are equally present in the genitourinary secretions of men and women with a wide range of urologic and gynecologic disorders, and studies of synovial fluids and membranes from patients with Reiter's Disease have only rarely revealed the presence of mycoplasmas (25, 27, 28). Furthermore, the arthritis they cause in animals is pyogenic in nature and both grossly and histologically differs from that of Reiter's Disease. Hence, in recent years it has been rather generally concluded that when present in these patients they are of merely secondary importance and are unlikely to be the primary causative agents. This question has been well reviewed by Sharp (29).

e) Virus: As in the case of any disease of unknown cause which has infectional features, it has been conjectured that Reiter's Disease may be viral in nature. This was suggested by Harkness in 1945 (30), who five years later reported finding inclusions in urethral and conjunctival cells (31). Others, however, have failed in attempts to isolate a virus (32, 33, 28). Currently, great interest has again been aroused in the possibility of a viral, or viral-like cause. In 1962 Siboulet and Galistin (34) reported viral inclusion bodies in urethral epithelial cells of three patients with Reiter's Disease. Elementary and initial bodies were found in chick embryos inoculated with material from these patients and eight of the twelve inoculated eggs showed malformations of the embryos. Three years later Dunlop and others (35) isolated the Tric agent, a member of the Bedsonia (or psittacosis/lymphogranuloma-venereum/trachoma/inclusion conjunctivitis) group, from urethral scrapings of several patients with non-gonococcal urethritis. More recently, Amor, Delbarre and Coste (36) have found inclusion bodies in urethral scrapings from 40 of 61 patients with Reiter's Disease (and in virtually 100% of the currently active cases) as well as in 7 of 30 patients with ankylosing spondylitis and involvement of peripheral joints, and in 53 of 135 patients with nongonococcal urethritis. They found similar inclusions in synovial cells from 10 of 17 men with Reiter's Disease. Still more recently, and working quite independently from the groups in Paris and London, Schachter and Engleman and their associates (37) have isolated Bedsonia from urethral and conjunctival scrapings and from synovial tissues from patients with Reiter's Disease, and serologic studies revealed increased complement fixation titers in 37%. It is to be noted that these isolations were obtained from inoculation of embryonated hen eggs, a method which had not been used in earlier attempts to isolate a virus (28). Although these important studies from Paris, London and San Francisco require further confirmation, it appears highly probable that, at long last, the cause of Reiter's Disease has been found, and that an agent of the Bedsonia group, either through its own toxic effects or by inducing a mechanism of the type discussed in the following paragraph, is the culprit.

f) Autoimmunity: In closing this discussion of causation of Reiter's Disease there remains the need to mention the possible role of autoimmunity or of delayed autosensitivity. If a mechanism of this type is to be invoked, and there is reason to suspect it, the "self plus X" theory of Lawrence (38) would appear best suited to the facts. According to this view, the intracellular presence of viable agents can lead to interactions between the cells and parasites which result in sufficiently foreign protein complexes to evoke self-destructive antibody responses. The fact that Bedsonia are obligatorily intracellular agents throughout the greatest part of their life within the host is in keeping with this type of immunologic mechanism.

In closing this lecture I wish to acknowledge again how privileged I feel to have been given this opportunity to take part in honoring Dr. Bach.

REFERENCES

1. Reiter, H.: Ueber eine bisher unerkannte Spirochateninfection (Spirochaetosis arthritica), Dtsch. Med. Wschr. **42:** 1535, 1916.
2. Fiessinger, N. J., and Leroy, E.: Contribution à l'étude d'une épidémie de dysenterie dans la Somme (Juillet–Octobre, 1916) Bull. et mém. Soc. méd. Hôp. Paris **40:** 2030, 1916–17.
3. Brodie, B. C.: *Disease of the Joints.* Longman, Hurst, Rees, Orme and Brown. London, 1818.
4. Manson-Bahr, P.: *The Dysenteric Disorders.* Cassel and Co. Ltd. London, 1943.
5. Huette: De l'arthrite dysentérique. Arch. Gén. de méd. **11:** 129, 1869.
6. Bauer, W., and Engleman, E. P.: A Syndrome of Unknown Etiology Characterized by Urethritis, Conjunctivitis and Arthritis (So-Called Reiter's Disease). Tr. Assoc. Am. Physicians **57:** 307, 1942.
7. Weinberger, H. J., and Bauer, W.: Diagnosis and Treatment of Reiter's Syndrome. Med. Clin. North America **39:** 587, 1955.
8. Ford, D. K.: Natural History of Arthritis Following Venereal Urethritis. Ann. Rheum. Dis. **12:** 177, 1953.
9. Hollander, J. L., Fogarty, C. W., Abrams, N. R., and Kydd, D. M.: Arthritis Resembling Reiter's Syndrome. J.A.M.A. **129:** 593, October 27, 1945.
10. Young, R. H., and McEwen, E. G.: Bacillary Dysentery as the Cause of Reiter's Syndrome. J.A.M.A. **134:** 1456, August 23, 1947.
11. Balboni, V. G.: Gonorrheal Arthritis, in Hollander and Collaborators, *Arthritis and Allied Conditions,* Fifth Edition, page 779, Lea and Febiger, Philadelphia. 1953.
12. Hench, P. S.: Arthritis. J.A.M.A. **132:** 974, 1946. Therapeutic "Information Please"
13. Ziff, M., Esserman, P., and McEwen, C.: Observations on the course and treatment of systemic lupus erythematosus. Arth. & Rheum. **1:** 332, 1958.
14. Shatin, H., Canizares, O., and Ladany, E.: Reiter's Syndrome and Keratosis Blennorrhagica. Arch. Derm. **81:** 551, 1960.
15. McEwen, C., Ziff, M., Carmel, P., DiTata, D., and Tanner, M.: The Relationship to Rheumatoid Arthritis of Its So-Called Variants. Arth. & Rheum. **1:** 481, 1958.
16. Cooper, N. S., and McEwen, C.: Unpublished data.
17. Good, A. E.: Involvement of the Back in Reiter's Syndrome. An. Int. Med. **57:** 44, 1962.
18. Olhagen, B.: Chronic Uro-polyarthritis in the Male. Acta. Med. Scandinav. **168:** 339, 1960.
19. McEwen, C., DiTata, D., Porrini, A., Good, A. E., and Rankin, T.: A Clinical and Radiologic Study of Ankylosing Spondylitis and Spondylitis Associated with Ulcerative Colitis, Psoriatic Arthritis and Reiter's Disease. To be published.
20. Wright, W., and Reed, W.: The Link Between Reiter's Syndrome and Psoriatic Arthritis. Ann. Rheum. Dis. **23:** 12, 1964.
21. Khan, M. Y., and Hall, W. H.: Progression of Reiter's Syndrome to Psoriatic Arthritis. Arch. Int. Med. **116:** 911, 1965.
22. McEwen, C., Michaelides, P., and Leibowitz, M. M.: Unpublished Observations.
23. Paronen, I.: Reiter's Disease. Acta. Med. Scandinav. Supplement 212, 1948.
24. Hess, E. V., Hunter, D. K., and Ziff, M.: Gonococcal Antibodies in Acute Arthritis. J.A.M.A., **191:** 531, 1965.
25. Dienes, L., Ropers, M. W., Smith, W. E., Madoff, S., and Bauer, W.: Medical Progress: Role of pleuro-pneumonia-like organisms in genitourinary and joint disease. New Eng. J. Med. **238:** 509, 503, 1948.
26. Dienes, L., and Edsall, G.: Observations on L-organism of Klieneberger. Proc. Soc. Exp. Biol. and Med. (N.Y.), **36:** 740, 1937.
27. Jonsson, J. quoted by Olhagen, B. See Reference 18.
28. Claus, G., McEwen, C., Brunner, T., and Tsamparlis, G.: Microbiological Studies of Reiter's Disease. Brit. J. Vener. Dis. **40:** 170, 1964.
29. Sharp, J. T.: The Mycoplasmataceae (PPLO) as Causes of Joint Infections. Arth. & Rheum. **7:** 437, 1964.
30. Harkness, A. H.: Cutaneous manifestations of gonorrhoea. Brit. J. Vener. Dis. **21:** 93, 1945.
31. Harkness, A. H.: *Non-Gonococcal Urethritis.* pp. 67, Livingstone, Edinburgh, 1950.
32. Ford, D. K.: Human tissue culture studies of non-gonococcal urethritis. Brit. J. Vener. Dis. **32:** 184, 1956.
33. Ford, D. K.: Further tissue culture studies on non-gonococcal urethritis in Reiter's Syndrome. Brit. J. Vener. Dis. **34:** 53, 1958.
34. Siboulet, A., and Galistin, P.: Arguments in Favour of a Virus Aetiology of Non-Gonococcal Urethritis Illustrated by Three Cases of Reiter's Disease. Brit. J. Vener. Dis. **38:** 209, 1962.
35. Dunlop, E. M., Al-Hussaini, M. K., Garland, J. A., Treharne, J., Harper, I., and Barrier, R. J.: Infection of Urethra by Tric Agent in Men Presenting because of "Non-Specific" Urethritis. Lancet 1: 1125, 1965.
36. Amor, B., Delbarre, F., and Coste, F.: Le

problème des inclusions cellulaires de nature virale dans certaines affections rhumatismales. Sem. Hôp. Paris **42:** 785, 1966.

37. Shachter, J., Barnes, M. G., Jones, J. P., Engleman, E. P., and Meyer, K. F.: Isolation of Bedsoniae from the Joints of Patients with Reiter's Syndrome. Proc. Soc. Exp. Biol. & Med. **122:** 283, 1966.
38. Lawrence, H. S.: The Relevance of Delayed Allergy and Homograft Sensitivity to Autoimmune Disease. Bull. Rheum. Dis. XII, No. 9, pp. 279, May 1962.

Hypersensitivity Reactions Involving the Lung[1]

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THE LUNG differs from most other organs in that antigens can reach it not only from the blood, but also directly from the atmosphere. Yet little is known with precision regarding this organ as a stage for immunological phenomena. It recent years the practice of luñg biopsy has added considerably to knowledge of lesions at the height of their development that would otherwise regress perhaps with only a nondescript residue of scar tissue. Still, at this time, a survey of pulmonary allergic reactions must be largely descriptive and even conjectural.

The presumption of an allergic pathogenesis is based on the presence of histological features commonly associated with immunological phenomena or dysproteinemias. These include, in the absence of other plausible explanations, 1. extensive eosinophilic reactions, 2. heavy infiltrations of plasma cells, 3. angiitis or granulomatosis or both, 4. non-caseating granulomas like those of sarcoidosis. The pulmonary manifestations of some allergic reactions were well known to Osler (1). A number of the microscopic changes were described by Klinge who as early as 1927 also suspected a controlling endocrine influence (2).

Relatively few attempts have been made to produce immunological pulmonary reactions in animals. Both horse serum (3, 4) and egg white have produced pulmonary lesions in sensitized animals (5). These were not accompanied by eosinophilia. The remarkable reactions in the lungs of rabbits injected intravenously with Freund's adjuvant are discussed later in relation to the pulmonary eosinophilias.

The main classes of presumably allergic pulmonary reactions in man can be conveniently categorized:

Putative Immunological Reactions in the Lungs

1. Exudative
2. Necrotizing Bronchitis
3. Asthma
4. "Pulmonary Eosinophilia"
5. Vasculitis
6. Collagen Diseases
7. Sarcoid-like Reactions
8. Others

In this grouping there is some overlap: 1. "Asthma" should be considered simply as a syndrome related to episodic bronchospasm. As such it can be observed without evidence of parenchymal disease. It can also continue, or even make its appearance, when pulmonary infiltrations and eosinophilia have become established. Asthmatic manifestations occur also in the course of some pulmonary angiitides and granulomatoses. 2. Minor vascular lesions have been noted in the acute exudative reactions, in the pulmonary eosinophilias, in primarily granulomatous disease (group 7), and in a miscellany of other conditions presumed tentatively to be allergic (group 8). 3. The occurrence of non-caseating

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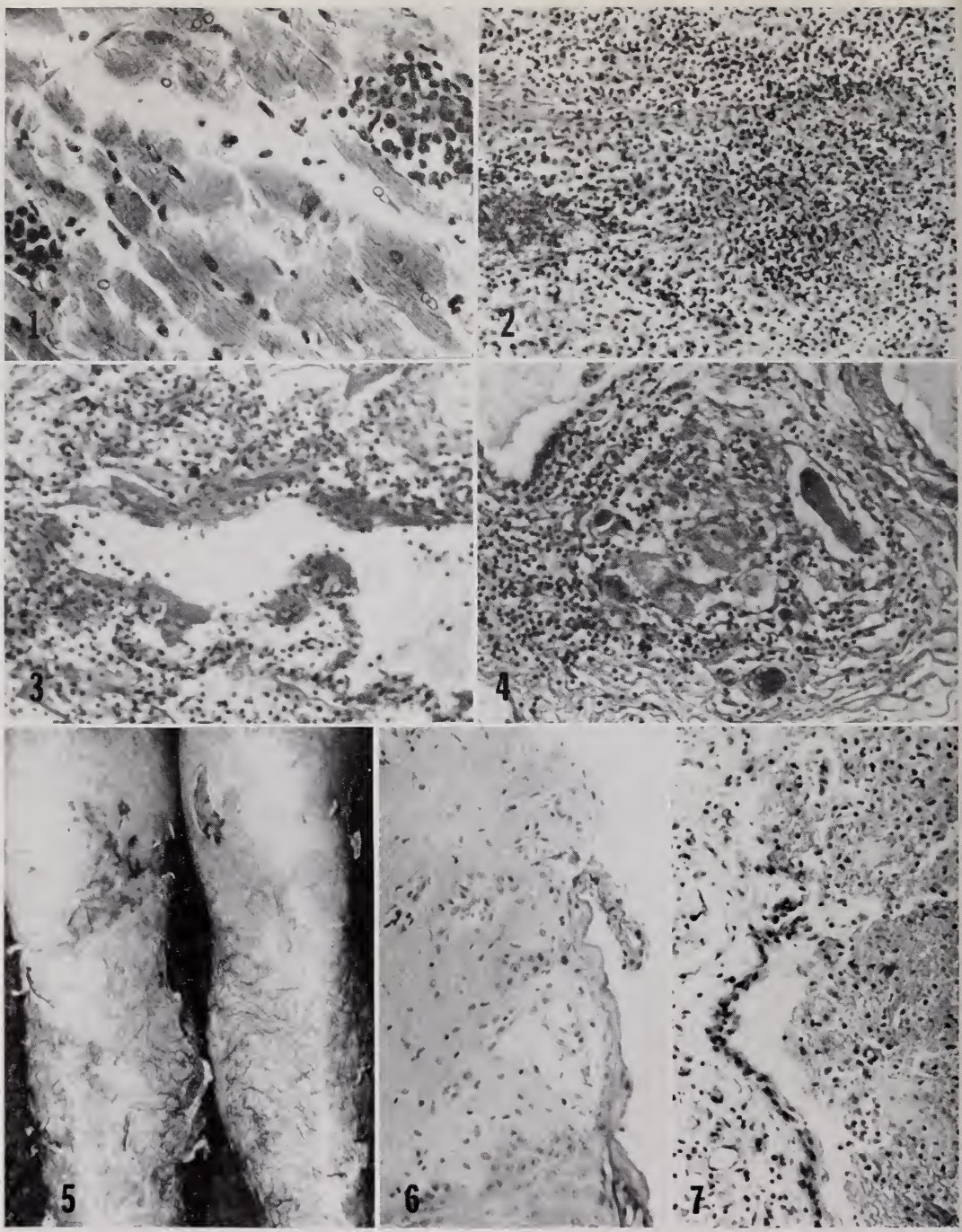


Figures 1–4. Generalized penicillin reaction; pre-existing sarcoid granuloma. The patient, a 63 year old woman, received penicillin and streptomycin for treatment of a foot infection thought to be streptococcal. Three days later she developed severe congestion of the conjunctiva. On the sixth day she had a migratory arthralgia and a leucocytic count of 16,200 with 34% monocytes. Death occurred on the eighteenth day after treatment was begun. Figure 1. Focal myocarditis. Necrosis of individual muscle fibers with predominately mononuclear infiltration. Figure 2. Spleen. "Trabeculitis." Fibrinoid change in collagen of a trabecula and heavy infiltrate of lymphocytes and plasma cells. Figure 3. "Diffuse alveolar damage" with formation of hyaline membranes and of a predominantly small mononuclear infiltrate. Figure 4. One of many granulomas of sarcoid type. The considerable hyalinization suggests that these antedate the acute exudative alveolar hypersensitivity reaction.

Figures 5–7. Penicillin reaction with exfoliative dermatitis and exfoliative bronchitis. The 25 year old man was given aqueous procaine penicillin G for an upper respiratory tract infection that had begun three days earlier. Penicillin had been given at least once in the past

granulomas like those of sarcoidosis in most of the other presumed sensitivity reactions is of particular interest. Indeed, in some it is reasonable to conclude that the non-caseating granulomas antedated the acute lesions of hypersensitivity. While none of these reactions is specific, all should serve as a stimulus to investigation of a possibly allergic state by all available methods.

Historically, the years 1931–1936 marked noteworthy advances, especially in clinical description of some of these entities. In 1931 Klinger (6) outlined, as a deviant form of periarteritis, the elements of the syndrome that now bears the name of Wegener (7), whose classical paper did not appear until five years later. In 1932 came the first clear description of Löffler's syndrome (8), of farmer's lung (9), and of maple bark disease (10), and in the same year, Bramwell and Ellis (11) suggested that allergy to components of cotton dust might be the mechanism of byssinosis. A case report illustrating a more severe and chronic form of pulmonary eosinophilia was published by Léon-Kindberg and associates (12) in 1940. Tropical eosinophilia was described by Frimodt-Möller and Barton (13) in the same year, and in greater detail by Weingarten (14) in 1943.

Exudative Reactions

Pulmonary edema may be the final event in anaphylactic reactions, but this may represent the consequence of physiological phenomena, rather than the direct effect of interaction of antigen and antibody. However, in some instances (for example, in certain reactions to penicillin), there is evidence of diffuse alveolar damage with hyaline membrane formation and a mononuclear interstitial infiltration resembling that in other tissues (Figs. 1–3). It is remarkable that lesions like those of sarcoidosis (Fig. 4) can sometimes be found in such acute allergic reactions, since these may themselves be indicative of an allergic state, as will be emphasized.

Exfoliative Bronchitis

Although exfoliative dermatitis occurring in reactions to such substances as penicillin is considered allergic, the precise pathogenesis of the lesion is obscure. Analogous lesions can occur in the bronchi (Figs. 5–7).

Asthma

Smooth muscle can function as an end organ in antigen-antibody reactions, as manifested in vasoreactivity or bronchospasm. These differ in various species. In persons dying in status asthmaticus, only rarely is there evidence that simple bronchospasm represents the mechanism of death. While some bronchi can appear constricted, in most instances the major effect is an actual distention of the small bronchi and bronchioles with large quantities of mucus, which grossly is remarkable for its highly viscous characteristics. Mucus-producing cells are present in excessive numbers in the glands and among the lining epithelial elements of bronchi and even within distal bronchioles. They also slough in large numbers. Basement membranes of bronchi and bronchioles are thick. Both of these changes also occur in chronic bronchitis uncomplicated by asthma. In the asthmatic syndrome, eosinophils and sometimes plasma cells, tend to be numerous and extend through all coats and even infiltrate in considerable numbers among the epithelial cells. Smooth muscle is often hypertrophied as well as hyperplastic, conditions that appear to be invariably associated when smooth muscle is subjected to increased tension. All of these changes are

without reaction, but on the day following the present treatment he developed a pruritic cutaneous erythema which became an exfoliative dermatitis (Figs. 5 and 6). Figure 7. Bronchus with necrosis of surface epithelium. The lesion resembles that of the skin in the scantiness of the cellular reaction. An exudate rich in protein and mononuclear cells fills the lumen.

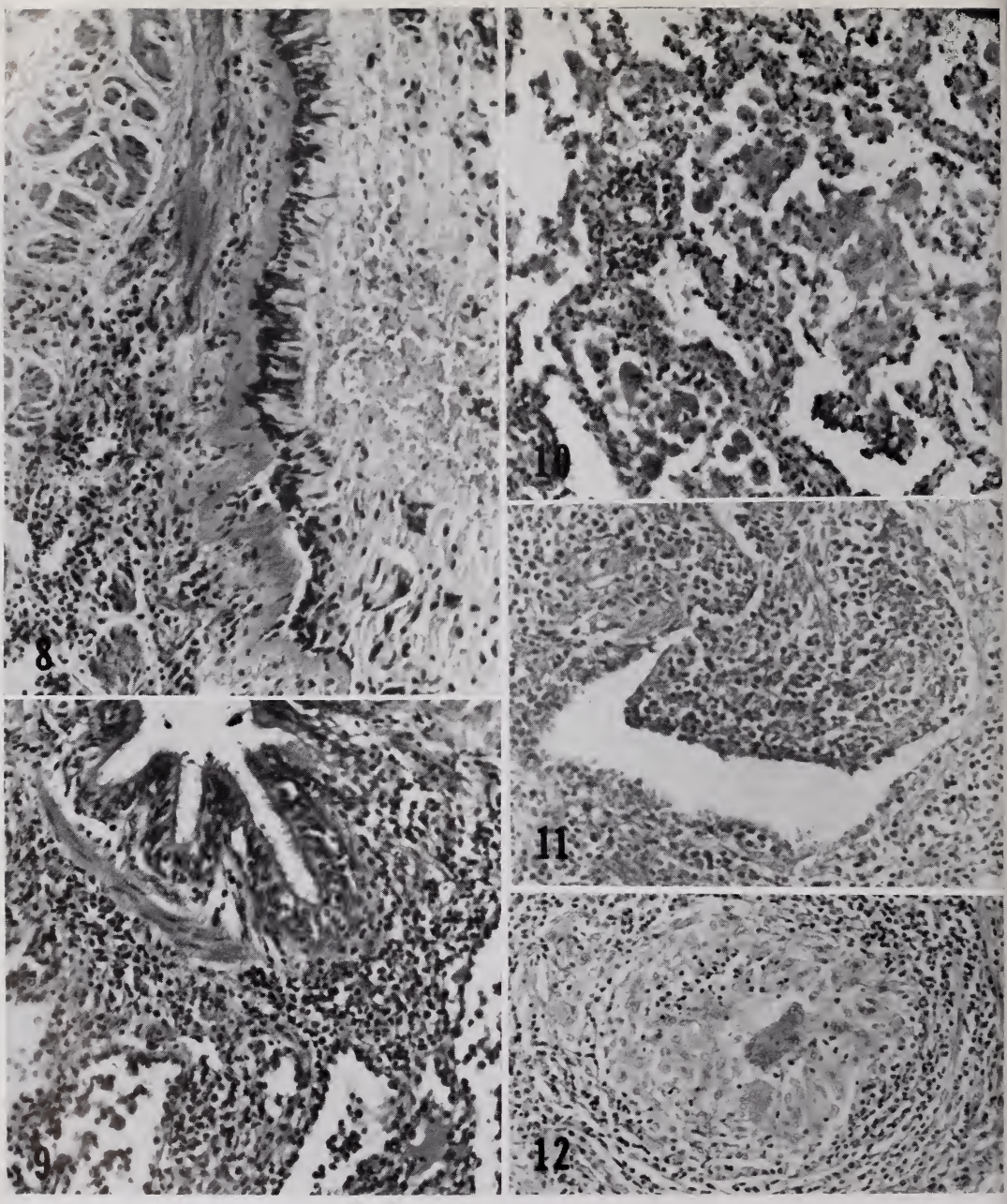


FIGURE 8. Sudden death in a 20 year old man with lesions like those of asthma, but without previous history. The lining epithelium of a large bronchiole is composed chiefly of mucus-producing cells, and a basophilic mucous plug fills the lumen. The basement membrane is thickened and the smooth muscle hypertrophied. All coats are infiltrated with lymphocytes, plasma cells and eosinophiles. The latter are present also within the mucous plug.

FIGURES 9, 10. Löffler's Syndrome. Patient was a 44 year old man with mild eosinophilia and pulmonary infiltration which cleared within three weeks on steroid therapy. Figure 9. Massive infiltration by eosinophiles of wall of bronchiole and of surrounding thickened interalveolar septa. The epithelium of the bronchiole is intact. Figure 10. The interalveolar septa contain numerous eosinophiles which are present also among the large mononuclear cells but fill the alveoli. This combination occurs commonly in the eosinophilic pneumonias. (See also Figs. 13–16.)

FIGURES 11, 12. Asthma complicated by pulmonary eosinophilia. Patient was a 47 year old physician who had been discharged from the army in 1944 because of asthma. Asthmatic attacks recurred until 1958. Pontocaine reaction occurred in 1958, and then the patient was well until 1964. In June 1964, he developed recurrent fever and cough, treated with broad-spectrum antibiotic. Multiple pulmonary infiltrations developed, especially of the right lower lobe. WBC was 10,000 to 14,000 with eosinophilia as high as 20%. Continuing cough produced

well known in fatal status asthmaticus (15, 16).

Less well known is that it is possible for an asthmatic attack to be fatal without previous history of the syndrome, although the typical lesions can be present. Asthma can be complicated by pulmonary infiltrations of various kinds. These can be ordinary pneumonias, but can also represent one class of the "pulmonary eosinophilias".

The Pulmonary Eosinophils

This designation is made purposely ambiguous, to suggest both pulmonary infiltrations associated with eosinophilia and eosinophilic infiltrations of the lung which may or may not be accompanied by an excess of eosinophils in the blood. Knowledge of the latter condition is owed to lung biopsies performed in search of explanation of pulmonary infiltrates. The term chosen is deliberately broader than "pulmonary infiltration and eosinophilia" as applied by Reeder and Goodrich (17). The classification proposed in the classical paper by Crofton and associates (18) is inclusive: 1. Simple pulmonary eosinophilia (Löffler's syndrome), 2. prolonged pulmonary eosinophilia, 3. tropical eosinophilia, 4. pulmonary eosinophilia with asthma, and 5. polyarteritis nodosa. This grouping is based on clinical criteria which have no specific etiological connotations. Thus, several of the syndromes, for example that of Löffler and the more prolonged pulmonary eosinophilia, were soon associated with ascariasis. However, other animal parasites such as filaria can produce the same manifestations which can persist over long intervals of time (18), and in the opinion of some students are responsible also for epidemics of tropical eosinophilia. Other helminthic parasites with like effects include hookworm, Strongyloides, Trichuris, Toxacara, Trichina, schistosomes, lung flukes, and ecchinococci (18, 19). Reactions to certain drugs can also be manifested in pulmonary eosinophilic infiltrations (20). Pepys and co-workers (21) suggested that hypersensitivity to *Aspergillus fumigatus* is related to some pulmonary eosinophilias and that the presence of precipitating antibody may mediate the production of arthus-like reactions in the lungs. Eosinophilia can accompany resolving pneumonic infiltrates in Hodgkin's disease, sarcoidosis and many other conditions, but when these are known to exist the designation Löffler's syndrome is, by custom, not employed. Rather, the major diagnosis is employed. It would seem as logical to adhere to this principle in designating according to etiology the pulmonary complications, direct or indirect, of the various helminthic infections.

In the subsequent paragraphs, Löffler's syndrome will be discussed because of its historical interest, yet inevitably the vague boundaries of the various entities named by Crofton (18) must become evident.

Löffler's Syndrome: The essence of the syndrome described by Löffler in 1932 is eosinophilia associated with wandering pulmonary infiltrations of brief duration, clinically silent or associated with minimal respiratory symptoms and fever (8). Recovery takes place within a month. A second series of 100 patients from Löffler's clinic was described by Maier (22) in 1943. Little is known about the pathological anatomy of this condition. The first description of the lesions is by von Meyenburg (23) who examined lung tissue from four patients who died violently or suddenly, without history or with minimal evidence of previous illness. The lesions

sanguineous sputum with no organisms on culture. Figure 11. Intruding into a bronchiole is a polypoid mass of granulation tissue heavily infiltrated with eosinophiles which extend also into the thickened interalveolar septa. The lumina of many alveoli are also filled with these cells. Figure 12. One of a number of granulomas similar to those of sarcoidosis which were scattered in the pulmonary parenchyma.

consisted of intra-alveolar, in some cases also interstitial, eosinophilic infiltrations together with serous and recently organizing fibrinous exudate. He also described giant cells formed of confluent proliferated alveolar cells. In two cases there was also a bronchitis and bronchiolitis with eosinophilic infiltrations and striking perivascular and perivenous accumulation of eosinophilic leukocytes. Von Meyenburg also described similar infiltrations of connective tissue in the liver. Two of the four patients had ascaris worms in the intestines, but larvae were not seen in the lungs.

Asthma Complicated by Pulmonary Eosinophilia: A number of rather diverse pulmonary lesions can complicate existing or recurrent bronchial asthma. In the experience of Saupe (24) some 11.6% of 355 persons with chronic asthma when followed over a period of 10 years developed nonspecific pulmonary infiltrations. Leopold has described a chronic "eosinophilic pneumonia" in asthmatics. The lesion is subpleural, particularly over the upper and anterior portions of the lung and tends to become organized, with some bronchiectasis and bronchiolectasis. Vasculitis can also occur in pulmonary eosinophilic infiltrations complicating asthma (25).

Rarely, persistent pneumonia in asthmatics can be associated with the development of intrabronchial polypoid masses of granulation tissue which are heavily infiltrated with eosinophils as are the walls of the distal air spaces. As in ordinary "bronchiolitis obliterans", an "obstructive" or "cholesterol" pneumonitis occurs distal to the bronchial polyps. Here, abundant fat can be demonstrated by appropriate staining. We have seen tissue derived by lung biopsy from two such patients. In one of these there were also scattered small sarcoid-like granulomas.

Of particular interest is a recent observation in a 44 year old woman with asthma beginning in childhood and with a count of 40% eosinophils in 9,000 leukocytes in the peripheral blood. In a lung biopsy the lesion closely resembled that of desquamative interstitial pneumonia (26) but with much more massive eosinophilic infiltration, not only in the septa of the lung, but also among the massively proliferated large mononuclear cells (macrophages, granular pneumocytes?) that filled the lumina of the alveoli. A remarkable process of transfer of the granules of eosinophils into the cytoplasm of the intra-alveolar large mononuclear cells was clearly in evidence, and these cells also contained brilliantly acidophilic crystalloids. Clinically there was remarkable improvement, with clearing of the lesions observed radiographically, after administration of steroids.

The pulmonary lesion found in this patient also is reminiscent of the remarkable proliferative reaction induced by giving complete Freund's adjuvant to rabbits intravenously. As demonstrated by Moore and co-workers (27, 28), one month after the first of three intravenous injections of the complete adjuvant masses of the large mononuclear cells pack the alveoli (29). Under the electron microscope the mononuclear elements comprise both alveolar lining cells and macrophages and the former are found to line the alveoli in greatly increased numbers. Remarkably, in rabbits this type of lesion subsides after 4–5 weeks without residual fibrosis. One interesting aspect of the experimental work, and also of the reaction in the patient just described, is the question that it raises of the possible immunogenic origin of desquamative interstitial pneumonia (26). The proliferative process in the lung of the rabbit in response to the adjuvant was already well developed before any specific immunological reaction was recognized by Moore and Schoenberg (28). In the present asthmatic patient the distribution of the lesion radiographically as a patchy infiltrate in the left upper lobe differs from

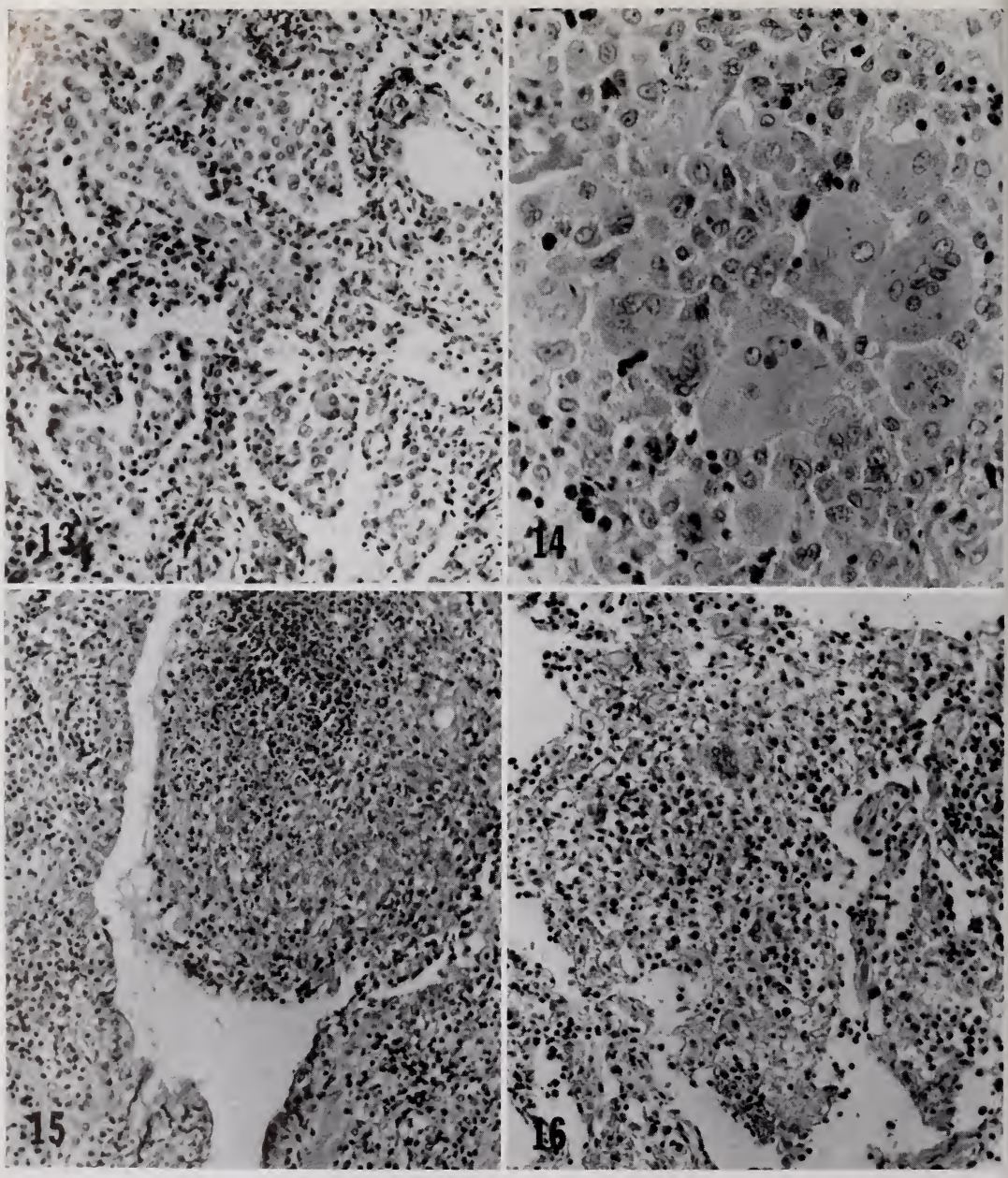
that usually found in desquamative interstitial pneumonia. Also, neither asthma nor even broncho-obstructive manifestations are a feature of that condition.

Chronic Forms of Pulmonary Eosinophilia: Mention has already been made of the non-specificity of the various syndromes and of the artificiality of the designation "Löffler's syndrome" on the basis of duration of the illness. The more chronic or recurrent syndromes are characterized by greater systemic manifestations such as high fever and malaise and by more signs and symptoms of pulmonary disease such as cough, chest pain, and sputum production. Rarely, pericarditis can accompany the pulmonary infiltrations (29). A number of these patients also develop asthma for the first time in association with the infiltrative pulmonary disease. While infections with helminthic parasites may be associated with such lesions, very high eosinophil counts (for example, 48% of 47,000 white blood cells and 67% of 28,000 white blood cells) can occur in man during the invasion stage of such worms as Ancylostoma and Strongyloides, with no signs of pulmonary disease (30). From wartime experience with epidemics of such infections, it seems probable that the actual symptoms indicative of pulmonary disease must be rare. It is also quite well established that worms need not necessarily be present in the pulmonary lesions when they exist and that these could represent distant allergic reactions. The same mechanism has been postulated for the angiitis often observed in trichinosis. Mites have also been found in sputum of persons with tropical eosinophilia (31). Most pulmonary infiltrations with eosinophils, however, have not been shown to be related to infections with animal parasites. Some of these seem to respond remarkably well to adrenocorticosteroid therapy, with subsidence of symptoms, pulmonary lesions, and pulmonary eosinophilia within two weeks (19, 32). Only antihelminthic therapy is effective, however, in certain parasitic infections. "Tropical eosinophilia" has been controlled by organic arsenicals. Two brief case reports will illustrate: a. A case of "prolonged pulmonary eosinophilia" of unknown cause, b. filarial infection with prolonged and recurrent disease responsive to carbamizine (33).

Eosinophilic Pulmonary Infiltrations without Peripheral Eosinophilia: Lesions typical of Löffler's syndrome as described by von Meyenburg (23) have been found in lung biopsies made for identification of obscure pulmonary infiltrates in the absence of blood eosinophilia. A typical example is illustrated in Figure 15. Here there were not only massive interstitial, especially perivascular, infiltrates with striking dilatation of the lymphatics, but bronchioles were also infiltrated and in part superficially ulcerated. Remarkable collections of loosely arranged epithelioid cells and small giant cells of Touton type have also been found as in the Löffler's lesions. These are not as circumscribed as the lesions of sarcoidosis, but the cellular elements involved appear to be of the same kind.

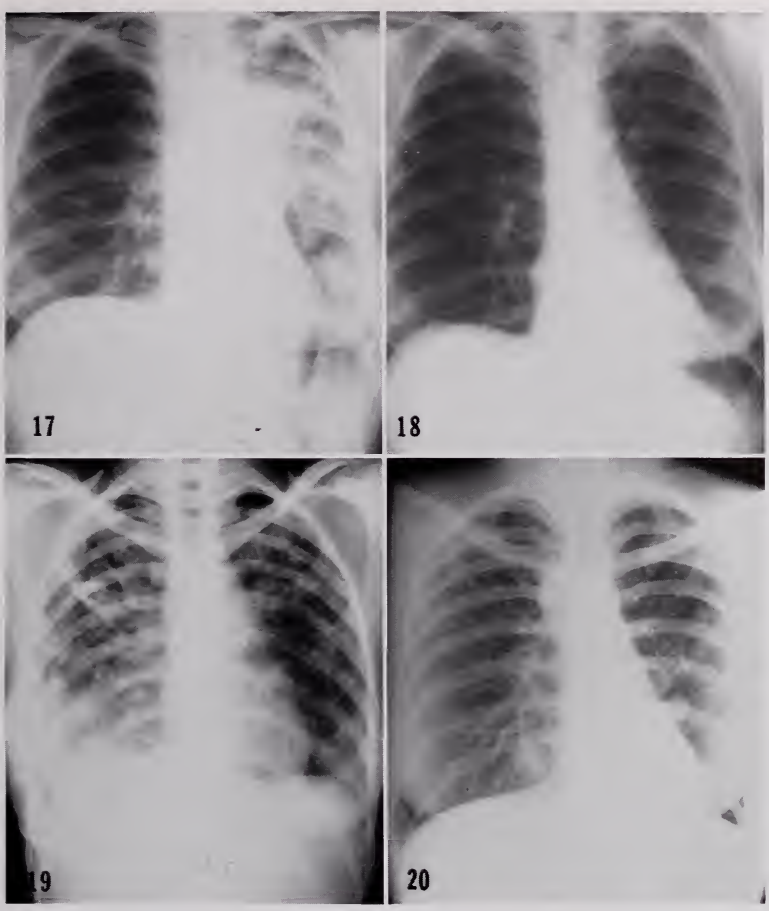
Vasculitis

The definition of vasculitis has varied greatly. Some use the term for simple infiltration of the adventitia with mononuclear cells, while others require evidence of damage to the wall, even necrosis. For present purposes "vasculitis" will be defined as infiltration at least of the media, or necrosis of the vessel. In this sense vasculitis can occur in minor degree in most types of sensitivity reactions and it may or may not be accompanied by eosinophilic infiltrations. There are a number of forms of pulmonary disease in which it is a prominent or dominant part of the reaction. It must be admitted that



FIGURES 13, 14. Prolonged pulmonary eosinophilia in asthma. (See also Figs. 17 and 18.) Numerous eosinophiles thicken the interalveolar septa and are mingled also with the abundant mononuclear cells which fill the lumina of the alveoli (Fig. 13). The reaction is similar to that in rabbits given Freund's adjuvant, except that in the latter eosinophiles are not so abundant. Figure 14. Granule transfer. Fragments of eosinophiles and isolated granules of the latter are present within the cytoplasm of giant cells. There are also brilliantly acidophilic crystals within some of the giant cells—minute Charcot-Leyden crystals. A large crystal is well seen within a giant cell at the left margin, center, of the photograph.

FIGURES 15, 16. Pulmonary eosinophilia without blood eosinophilia. Patient was a 34 year old woman with persistent cough following an influenza-like illness that had begun in December 1962. Infiltration of the right middle lobe and dilatation of bronchi led to a right middle lobectomy in May 1963. No blood eosinophilia occurred at any time. Masses of eosinophiles and large mononuclear cells within a proteinaceous exudate. There is necrosis of bronchioles with intrusion of organizing exudate into the lumen (Fig. 15). A Touton giant cell is seen in the center of a plug of exudate that fills the lumen of a distal air space in Fig. 16.



FIGURES 17, 18. Prolonged pulmonary eosinophilia complicating asthma. Same patient as in Figs. 13 and 14. Remarkable clearing of infiltrative process from Jan. 1965 (Fig. 17) to Mar. 1966 (Fig. 18) after institution of steroid therapy.

FIGURE 19. Allergic non-eosinophilic pneumonia. Same patient as in Figs. 21–24. There are disseminated minute vaguely defined densities. Partial clearing of these infiltrates occurred slowly after several months of steroid therapy but with little relief of malaise, dyspnea and fever, which were the chief manifestations of the illness. No blood eosinophilia was detected.

FIGURE 20. "Limited Wegener's granulomatosis." The lesions resemble those of a disseminated neoplasm. They are typically multiple, bilateral, predominate in the lower lobes, and some have cavitated.

the relationship to hypersensitivity or other phenomena of immunity is still speculative and not supported by direct evidence. As a basis for discussion the following classification of angiitides will be employed:

Angiitides Involving the Lung

A. Bronchial arteries
   1. Polyarteritis nodosa

B. Pulmonary vessels
   1. Polyarteritis nodosa
   2. Wegener's granulomatosis
      a. Usual
      b. "Limited"
   3. Churg and Strauss: "allergic granulomatosis"
   4. Fienberg: "Pathergic granulomatosis"
   5. Others

The bronchial arteries can be involved in the course of polyarteritis in its usual clinical manifestations (34), with or without blood eosinophilia. Disseminated nodular lesions can then sometimes be seen radiographically. This condition will not be discussed in detail.

Pneumonias with perivascular and intra-alveolar infiltrates resembling those of Löffler's pneumonia, but without excessive numbers of eosinophils in the peripheral blood or in the lesions also occur in the lung. The cellular population is a mixture, with a predominance of polymorphonuclear leukocytes but with numerous foamy large mononuclear cells, lymphocytes and plasma cells as well. Fibrinoid changes in vessels, especially small venules, can occur very widely through the lung. Thrombosis complicates the process and small infarcts, sometimes hemorrhagic and sometimes "white", complicate the lesions. Their allergic origin is postulated simply on histological criteria without other evidence.

The designation "Wegener's granulomatosis" is now commonly applied to a syndrome associated with angiitis and focal necrosis of tissue with granulomatous reaction beginning in the respiratory tract, spreading in varying degrees to other tissues and terminating in a focal necrotizing glomerulonephritis similar to that of Henoch-Schönlein purpura (35). We have now observed more than 30 patients with pulmonary lesions identical to those of Wegener's granulomatosis, but with absent or limited lesions elsewhere, and without evidence of glomerulonephritis (36). One patient in this group has survived for more than 13 years, despite some dissemination of the process. There are some similarities but also distinct differences between this condition and the "allergic granulomatosis and angiitis" described by Churg and Strauss (37). In the latter, organs other than the lung, and especially the spleen and heart, are involved and there tends to be extensive eosinophilic infiltration of the tissues and also blood eosinophilia. The course can be extremely rapid (38).

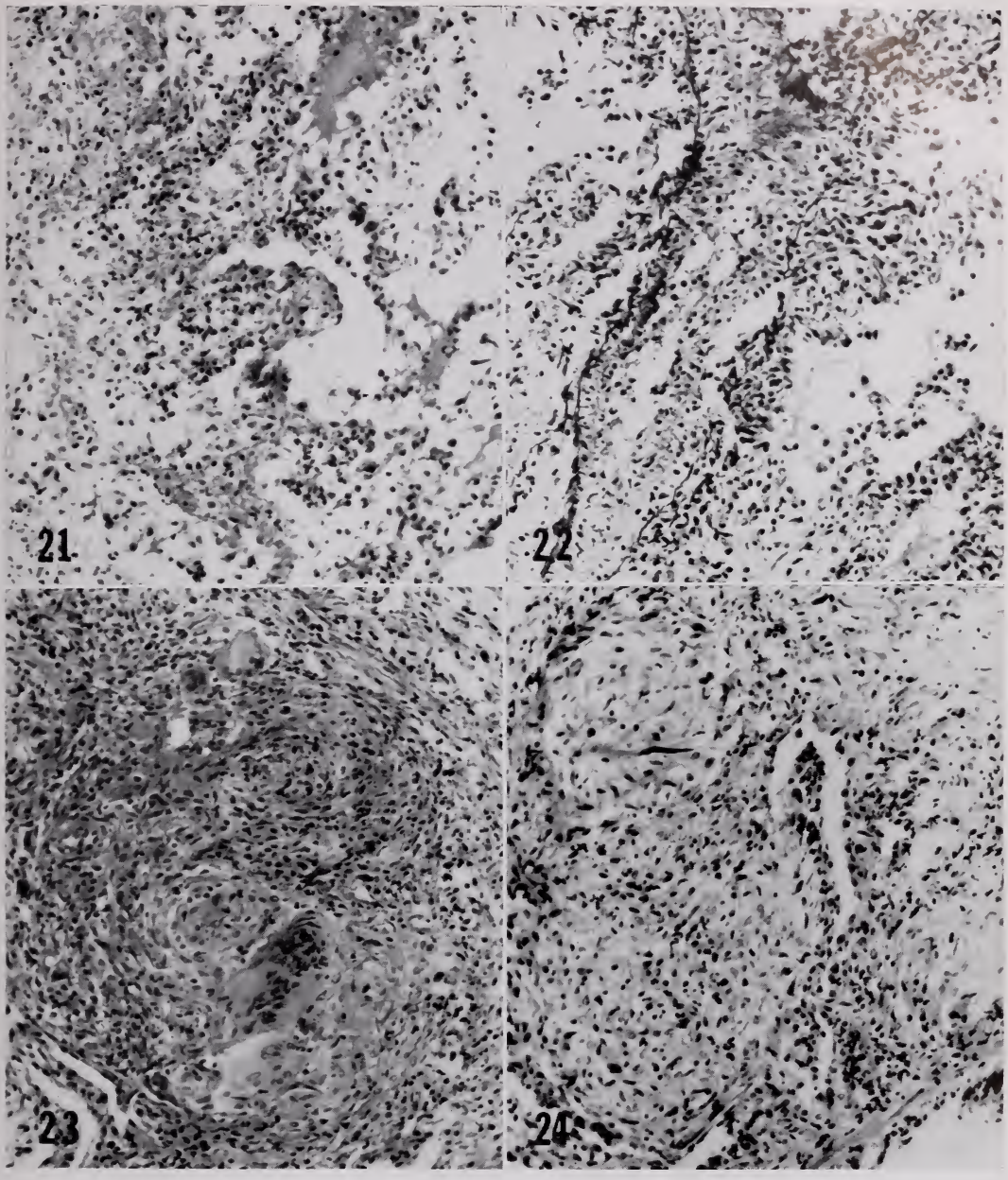
In the limited forms of Wegener's granulomatosis, symptoms can be vague or absent despite massive intrapulmonary lesions. Most patients, however, have malaise, cough, fever, dyspnea and weight loss. Pleural pain occurs in less than half of the cases, and effusions in less than 20%. A leukocytosis is often present, almost always without eosinophilia. Radiographically there are usually multiple lesions which predominate in the lower lobes, and which are only rarely present in the uppermost portion of the upper lobes. Approximately one-third are visibly cavitated in the roentgenograms. Lung tumor is the working diagnosis in almost all. Anatomically the lesions resemble white infarcts or caseous or organizing granulomas. Angiitis, especially granulomatous angiitis, involving both arteries and veins and especially the latter, which is manifest outside of the foci of necrosis as well as within them, is the clue to the diagnosis. These changes should be sought as diligently in any "granuloma of unknown etiology", as would be the usual infectious agents. When renal lesions have occurred in this group, they have been in the form of large tuberculoid granulomas similar to those of the lungs, and not the focal glomerular lesions.

In the syndrome discussed by Fienberg (39, 40) as "pathergic granulomatosis", a nodular granulomatosis and angiitis similar to that just described and at least for several years limited to the lung is associated with asthma and eosinophilia.

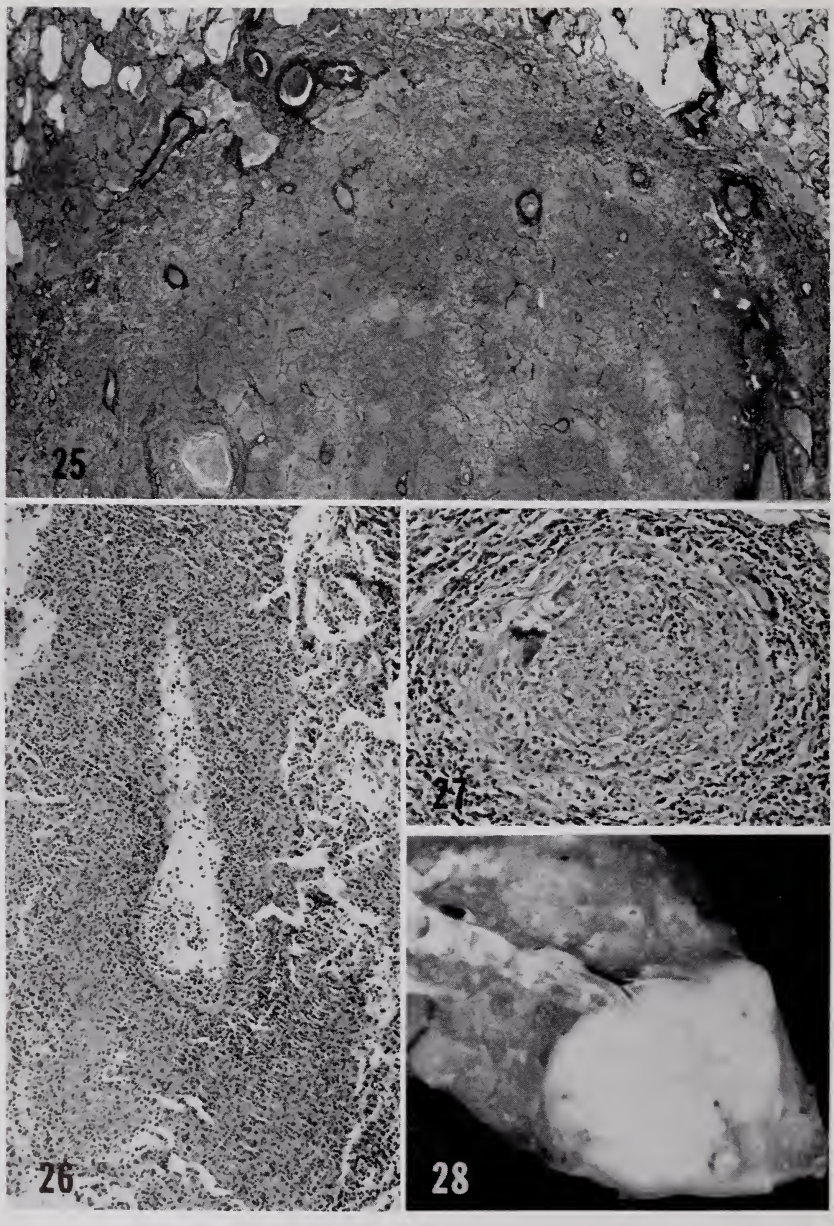
Collagen Diseases

In collagen diseases other than vasculitis, the lungs can be involved in a variety of ways, for the most part non-specific. Rather characteristic lesions occur in some patients with lupus and rheumatoid disease.

In lupus erythematosus there can occur



FIGURES 21–24. Allergic pneumonia without eosinophilia in the same patient as in Fig. 19. The process is in part exudative with proteinaceous material embedding scattered polymorphonuclear leucocytes, lymphocytes and plasma cells. Eosinophiles are rare (Fig. 21). There is an angiitis and periangiitis with fibrinoid necrosis and obliteration of lumen as exemplified in the venule illustrated in Fig. 22. Minute non-caseating granulomas are widely scattered throughout the tissue (Fig. 23) and some have become partially organized (Fig. 24).



FIGURES 25–28. "Limited Wegener's granulomatosis." The lesion resembles a "white infarct" but has become rounded, similar to an infectious granuloma (Fig. 25). Numerous pulmonary arteries and veins both within and outside of the lesion show changes similar to those illustrated in Fig. 26. Here an artery has become necrotic and is heavily infiltrated with an exudate predominantly of large and small mononuclear cells, including plasma cells. Eosinophiles are very scanty. There is also necrosis of the surrounding tissue, and bronchi can be involved with similar infiltration. This process represents "granulomatosis". It accounts for much of the change demonstrated in Fig. 25 which cannot be ascribed simply to infarction. Noncaseating granulomas resembling those of sarcoidosis (Fig. 27) occur in one-third of the cases. Grossly the lesion resembles a white infarct or granuloma (Fig. 28) where it is centered upon an artery but also envelopes the associated bronchus.

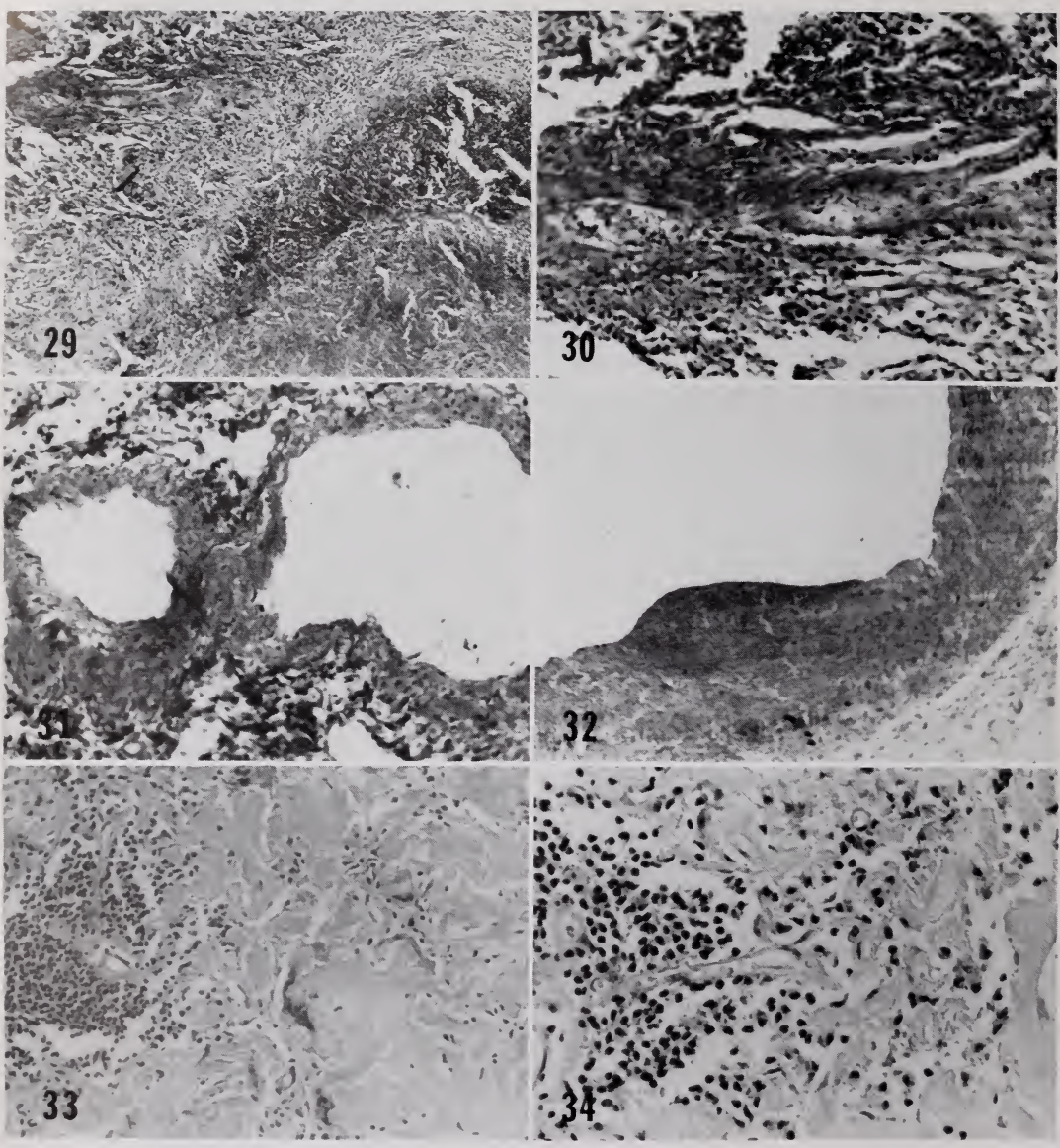
an alveolitis with fibrinoid change involving the entire structure, including the small vessels (41, 42). There are not only acidophilic hyaline membranes, but structurally similar masses of material become hematoxylinophilic, in part from incorporation of the substance of nuclei in process of necrobiosis. Vasculitis and thrombosis of larger vessels can also be found (43).

Lesions of the lung related to rheumatoid disease are of four main types: 1. Interstitial pneumonia, 2. granulomatous lesions, 3. vascular lesions, and 4. amyloidosis. The pulmonary lesion most commonly associated with rheumatoid arthritis is an interstitial pneumonia, but it is, in fact, a rare complication. Doctor and Snider (44), however, state that 20% of persons with diffuse interstitial pneumonia have rheumatoid arthritis. In their fully developed state the lesions cannot be distinguished from most other interstitial pneumonias. They can progress ultimately to "honeycombing", as with other "collagen diseases", for example diffuse sclerosis (45). In all of these the honeycombing tends to be most extensive in the lower posterior portions of the lobes, but involves also the anterior parts of the lingula and middle lobe. A clue to the nature of the process was obtained from a patient who had had clinical and radiographic evidence of interstitial pneumonia for more than five years who died suddenly of subluxation of the cranium on the spine. Although most of the changes were quiescent, there were small foci of alveolitis with fibrinous membranes, and an infiltrative and exudative lesion in which large and small mononuclear cells and plasma cells predominated, with a remarkable resemblance to the usual articular and periarticular lesions. This observation suggests that the process in the lungs can be slowly and subtly progressive, as in the joints. There is a slow breakdown of the tissue without overt signs of pneumonia.

The granulomatous lesions when present can be in every detail replicas of subcutaneous nodules. Here a layer of palisaded epithelioid cells is arranged about a central core of necrotic, fibrinoid or hyaline material. The last mentioned, however, may be enclosed simply by a capsule of connective tissue infiltrated with variable numbers of plasma cells. Cavitation is sometimes observed. Pneumoconiosis associated with rheumatoid disease is characterized by large confluent granulomas within which crystalline silicates can be demonstrated—an association that has come to be known as Caplan's syndrome (46–48). Clinically the massive agmination of the lesions of pneumoconiosis should suggest the existence of rheumatoid arthritis.

In rheumatoid disease vasculitis often accompanies the granulomata. In some the vascular lesion is merely a collaring or infiltration with small mononuclear cells and especially plasma cells, and the lumen in such vessels is sometimes restricted or even subtotally occluded by fibrous connective tissue. Often the changes are acute and of fibrinous type which is rare in Wegener's granulomatosis. The cellular exudate in rheumatoid disease also is less variegated. The most remarkable of the vascular changes is a laminar and massive fibrinoid transformation of the entire wall, in some instances with "onion skin" lamination (49). This can lead to aneurysmal expansion of considerable magnitude, in some instances large enough to be visible radiographically. Thrombi or additional deposits of amorphous protein form in such vessels which are partly occlusive and can give the appearance of a cavitative lesion.

Nodular pulmonary amyloidosis can also be an accompaniment of the rheumatoid state. Among the massive fissured deposits



FIGURES 29 TO 34.

FIGURES 29, 30. Rheumatoid nodule in lung with associated angiitis. The center of the nodule shown in Fig. 29 is partially hyalinized. Externally there is more basophilic material with fibrinoid change, and necrotic polymorphonuclear leucocytes are present here in large numbers. In the capsule of granulation tissue are numerous plasma cells. Palisaded epithelioid cells, often found in pulmonary as well as subcutaneous rheumatoid nodules, are not present. In the upper left of the photograph is a vessel with fibrinoid necrosis, shown at higher magnification in Fig. 30. The lumen is filled with protein enclosing polymorphonuclear leucocytes. The lesions shown in Figs. 29 and 30 resemble those of "limited Wegener's granulomatosis" but differ in that they show extensive fibrinoid change.

FIGURES 31, 32. Fibrinoid change in wall of pulmonary artery with aneurysmal expansion. In the vessel at the left, which spirals through the field (Fig. 31), only a few muscle cells are visible and refractile fibrinoid material composes most of the wall. A layer of cellular fibrous

can be seen groups of plasma cells and Marschalko giant cells and adjacent blood vessels are often heavily infiltrated by the former.

Pulmonary changes in other collagen diseases will not be discussed in detail. In his general discussion of pulmonary changes in collagen diseases, including rheumatic fever, dermatomyositis and scleroderma, Kuzma (41) theorized that a common basic feature was the precipitation of acid mucopolysaccharides. While substances of this class are deposited in these conditions, this must be only a part of a much more complex process. The precise mechanisms of damage to tissue remain to be elucidated. In rheumatic fever many of the lesions that have been ascribed to "rheumatic pneumonia" probably represent the effects of extreme pulmonary congestion or of pulmonary hypertension (50), although the direct effect of the "rheumatic process" is still moot. In scleroderma, lesions leading to honeycombing are not rarely associated with peripheral pulmonary neoplasms (45).

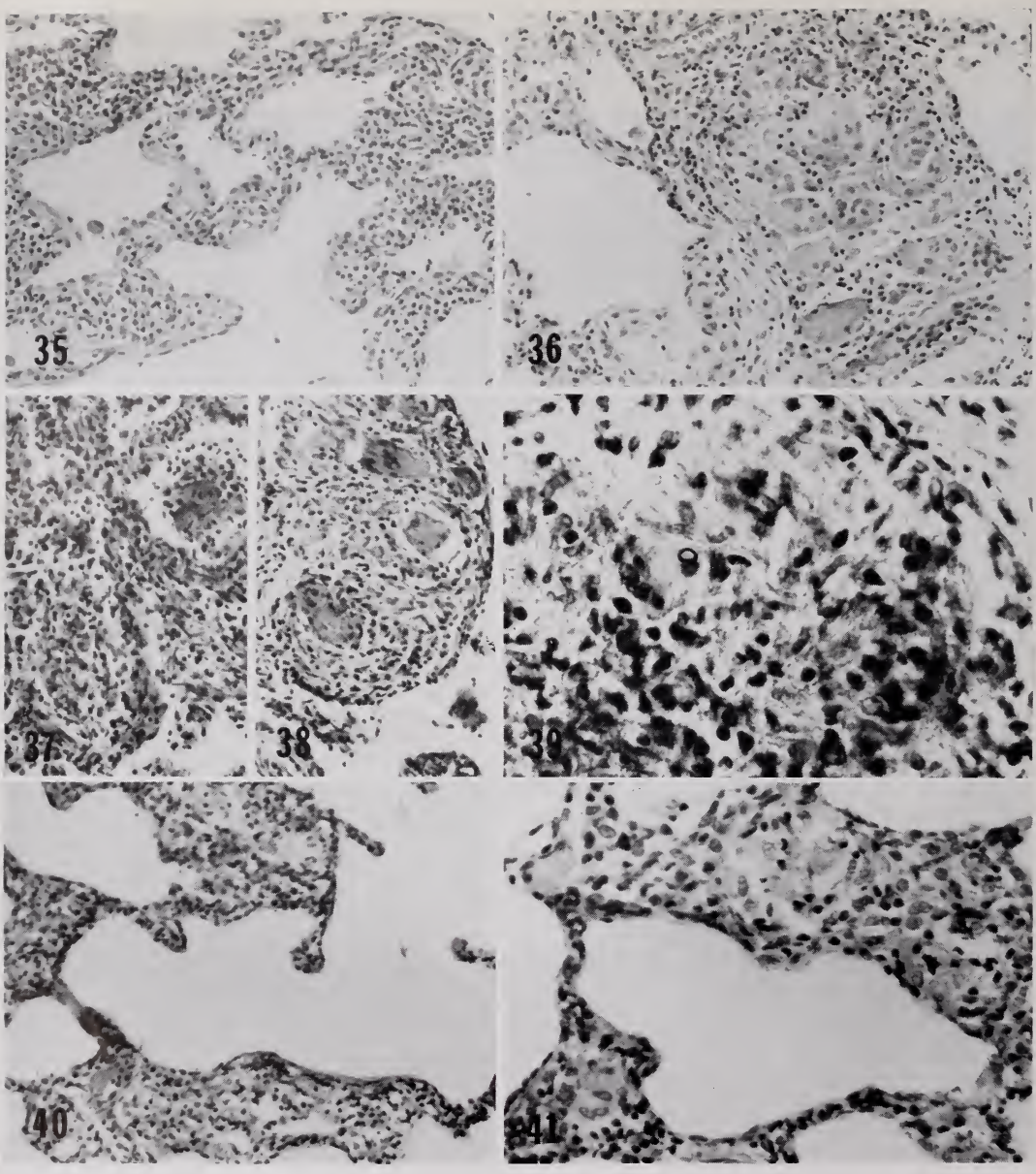
Granulomatous Lesions Resembling Those of Sarcoidosis

Excellent evidence has now accrued that granulomatous lesions like those of sarcoidosis can represent an important type of reaction to a number of antigenic substances present in nature. In fact the presence of such non-caseating granulomas should arouse the suspicion of an allergic factor in the tissue response.

One group of such reactions, that occur under appropriate conditions of exposure followed by a characteristic symptom complex, has been designated "farmer's lung" (9, 51–53). Within a few hours after exposure to moldy organic dusts such as those of stale hay, sensitive subjects develop chills, fever, cough and dyspnea. The physiological disturbance is manifested in a decrease in vital capacity, especially inspiratory capacity, and diminution of the maximal expiratory and inspiratory flow rates. Evidences of air-trapping are rare. Signs of "alveolo-capillary block" are present, with evidence of an abnormality in gas distribution within the lungs (51). Radiographically the appearances are those of a diffuse interstitial pneumonia with fine nodular densities especially in the lower and lateral portions of the lungs. Descriptions of the histological appearances vary depending on the time when tissue is obtained in relation to exposure. In the experience of Emanuel (52) who saw the lesions relatively early, they appear to be interstitial with a predominance of lymphocytes, but with large numbers of plasma cells as well, together with occasional neutrophils and eosinophils. Large histiocytes with foamy cytoplasm are also present, but well-defined non-caseating granulomas with Langhans cells were found only in 9 of 24 biopsies. Emanuel found foreign material, sometimes doubly refractile, in more than half of the biopsies. Rankin and coworkers (51) on the basis of tissue obtained in later stages found more of the non-caseating granulomas that would be indeed difficult to distinguish from those of sarcoidosis or berylliosis. They found the physiological disturbances and symptoms

connective tissue lies in sub-intimal position at the left. In Fig. 32, a segment of a much larger aneurysm of the pulmonary artery is seen. Here there is a layer of fibrinoid material much thicker than the original wall. Such aneurysmal transformation, which can produce large shadows in the roentgen film, represents another pathogenesis in the formation of one type of "rheumatoid nodule" in the lung.

Figures 33, 34. Nodular amyloidosis complicating severe rheumatoid arthritis. The deposits were visible as well defined somewhat irregular rounded shadows in the roentgenograms. Giant cells are present in the interstices among the amyloid deposits (Fig. 33), where there are also scattered plasma cells. The latter tend to aggregate about the partly obliterated residua of small vessels (Fig. 34).



Figures 35, 36. Farmer's lung. Infiltration of septa among distal air spaces by lymphocytes and plasma cells (Fig. 35). Non-caseating granuloma similar to that of sarcoidosis (Fig. 36).

Figures 37–39. Maple bark disease. Three fields from the same fragment of lung obtained at biopsy. Figure 37 demonstrates an exudative component, probably the early phase of the reaction. Masses of deeply staining protein enclose small mononuclear cells which also infiltrate the interalveolar septa. Elsewhere much of the lesion resembles that of Fig. 35. In Figure 38 is shown a typical non-caseating granuloma. To the left and above the center of the photograph in Figure 39 is seen a thick-walled refractile spore of *Cryptostroma corticale*. The outer wall appears brown in unstained preparations, a color retained despite staining of the sections with hematoxylin and eosin. The spore lies within a giant cell near the center of a non-caseating granuloma.

Figures 40, 41. Pigeon-breeders' disease. In Figure 40, the tissue response is exquisitely interstitial and consists of an infiltrate of lymphocytes and plasma cells together with epithelioid cells which tend to be aggregated. In other foci, the small mononuclear cells infiltrate the walls of bronchioles and fill the lumina of some distal air spaces, embedded in proteinaceous material. Characteristic minute epithelioid granulomata are seen in Figure 41. These contain abundant fat.

to be reversible with a corresponding clearing of the chest film. Repeated exposures over a period of time, especially in certain individuals were found, however, to lead to irreversible fibrosis and even death from pulmonary insufficiency.

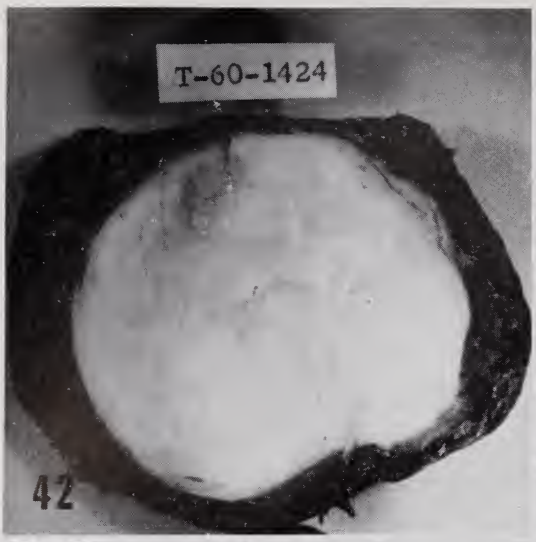
The demonstration of precipitins against extracts of moldy hay, and more specifically against hay antigen prepared by inoculation of the sterilized material with the actinomycete *Thermopolyspora polyspora,* suggest hypersensitivity to certain of these agents. This is confirmed by the effects of inhalation of the extracts by susceptible persons (54). Immunological phenomena are discussed in detail also by Bishop *et al.* (53).

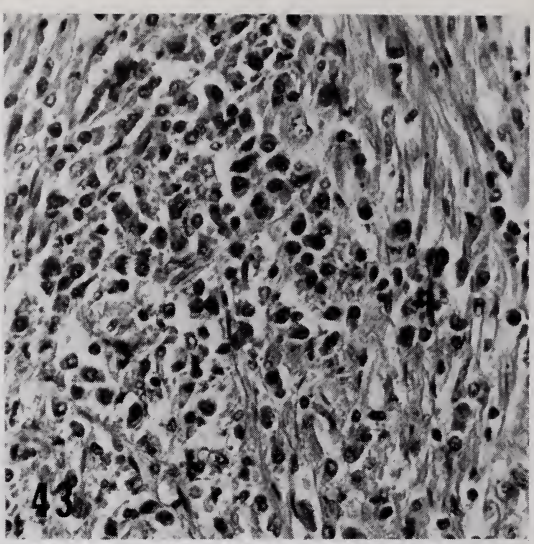
What has been called "maple bark disease" (10) has many analogies to farmer's lung clinically, radiographically, and in the appearance of the non-caseating granulomatous lesions in the lung (55). These contain the spores of the fungus *Cryptostroma* (*Coniosporium*) *corticale* which is responsible for sooty bark disease of certain maples (56). In human tissues the spores and bits of mycelium have distinctive thick brown walls which reduce silver in the Grocott stain. The organisms do not grow in the tissues, but persist as foreign bodies. They can be easily grown, however, from the tissues at room temperature. Intracutaneous inoculation with protein extract of the spores in affected individuals produces an immediate wheal which develops a hemorrhagic center surrounded by a broad zone of erythema. A protein extract of the organisms is competent to sensitize guinea pigs.

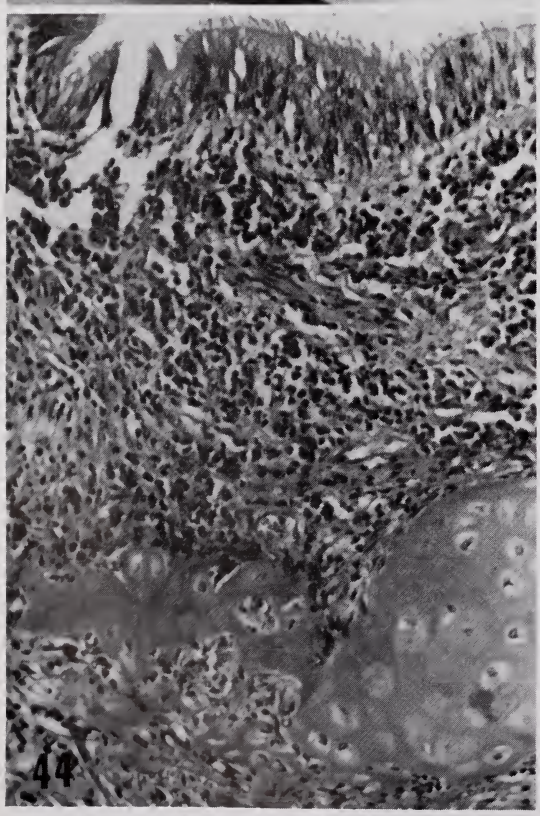
A remarkable group of essentially similar reactions in terms of clinical symptomatology have also been observed in pigeon handlers, and also among other bird fanciers exposed to dust from cages and feathers (57). As in farmer's lung, shortness of breath and cough develop in 4–8 hours after exposure and these may be accompanied by fever, chills, night sweats, and myalgia that can recur episodically. Fine diffuse pulmonary infiltrates are visible radiographically. Transient eosinophilia can be present which tends to clear very rapidly. Symptoms can be induced by a "challenge" with dust from pigeon feathers, or by the injection of sterile pigeon serum. Sera of patients give a positive precipitin reaction by immunodiffusion with pigeon serum. The presence of precipitating antibodies, however, does not indicate existence of clinical disease.

Undoubtedly, similar reactions to other material in the environment will be brought to light. Allergic reactions have been suspected in persons engaged in mushroom growing but have not been established, and indeed it is possible that these result from exposure to noxious gases. Oxides of nitrogen have been under suspicion in this regard (58).

Byssinosis is a symptom complex that occurs in cotton workers, manifested as a recurrent feeling of tightness in the chest and dyspnea especially on re-exposure after an interval (59). Antibodies to certain antigens of the cotton plant, especially the receptacle of the flower, have been demonstrated (60). The antibody levels are highest in the serum of the cardroom workers and have been found in lesser concentrations in exposed individuals without byssinosis. Desensitization appears to be ineffective in preventing the recurrent syndrome (59). The suggestion has been made on the basis of experiments in animals that certain parts of the plant when introduced into the tissue may function as histamine liberators, similar for example to the substance 48–80 (61). Such physiological investigations may provide important clues to the pathogenesis of these conditions. The status of byssinosis as an allergic reaction is somewhat in doubt at this time.







FIGURES 42, 43. Plasma cell granuloma. A large ovoid mass of light tan color bulges above the cut surface of the lung from which it appears to be sharply delimited (Fig. 42). It is, however, unencapsulated and plasma cells infiltrate into the surrounding interalveolar septa and bronchioles. The lesion consists of masses of plasma cells among which are bundles of elongated fibroblasts. Some of the plasma cells also assume an elongated appearance (Fig. 43).

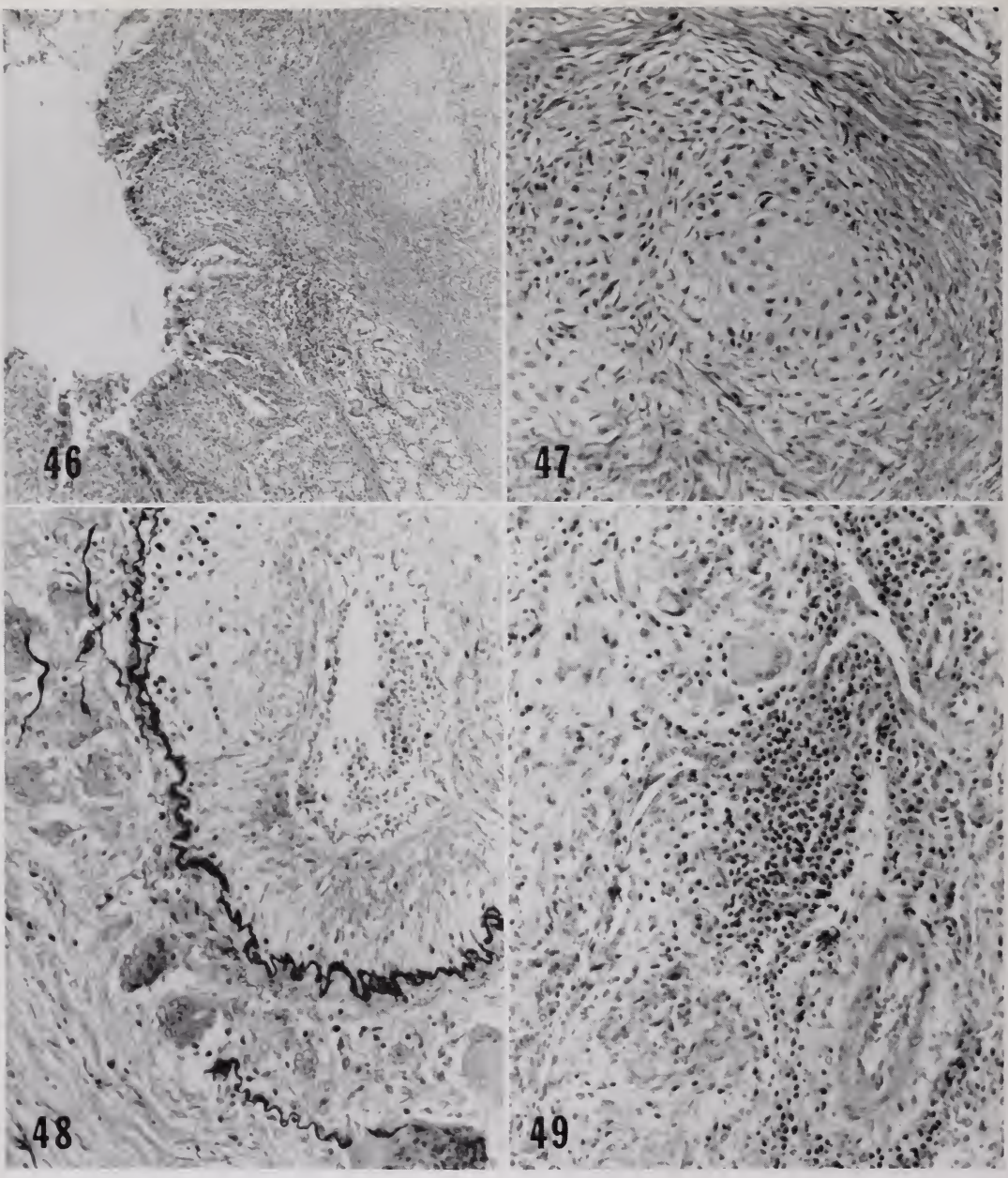
FIGURES 44, 45. Chondritis with collapse of bronchi—auto-immune reaction to cartilage? The patient was a young physician in the air corps with recurrent attacks of pulmonary disease and massive atelectases and infiltrations. The perichondrium has become infiltrated with masses of plasma cells which occur also within the denuded cartilage, even among the chondrocytes. Cartilage has become resorbed, to the point of disappearance in some foci. Small granulomas, like those of sarcoidosis, were in the parenchyma elsewhere.

Miscellaneous Lesions Possibly Related to Immunity Phenomena or Dysproteinemia

Plasma Cell Granulomas: Among the common tumor-like lesions of the lung, there occur more-or-less circumscribed but non-encapsulated masses of plasma cells admixed in varying degrees with other leukocytes. That these must be quite common is suggested by the fact that we have seen no less than 30 examples. They have often been mistaken for malignant plasmacytomas or myelomas. In our experience, however, these have all proved to be confined to the lung, unassociated with the biochemical changes of myelomas, and without tendency to recurrence after even limited resection (62). In these lesions the plasma cells for the most part are mature, and Russell bodies may be common. On occasion nests of larger cells suggest germinal centers in their arrangement. These consist in part of large reticuloendothelial cells, but in part also of cells transitional to mature plasma cells. More elongated spindle-shaped cells in bundles and whorls can be prominent in some portions of these lesions. This has led on occasion to a mistaken impression of Schwannoma. While for the most part these cells suggest fibroblasts, their nuclei often have plasmacytoid characteristics. Occasionally there are groups of lymphocytes, histiocytes and fat-filled cells, some with histiocytic and some with plasmacytoid characteristics. While certain of these lesions have been called "inflammatory pseudo-tumors", we prefer the designation plasma cell granuloma. They are often asymptomatic and discovered simply on routine radiographic examinations. Some patients have symptoms of cough and other evidences of pulmonary disease. They can occur even in early childhood. One typical example was discovered during a pre-school examination in a 6 year old child by a physician who found an unexplained dullness over the left lower lobe. Subsequently radiographic examination disclosed a large, rounded tumor-like mass, which was removed by lobectomy. Bronchi are sometimes surrounded by the lesion, as if entrapped. Occasionally necrosis occurs near the centers of the lesions. We have seen one example of a plasma cell granuloma in a major bronchus in a child where it produced evidences of obstruction. None of these lesions has recurred even after local resection. In a few patients electrophoresis has not revealed abnormalities of the blood protein pattern.

Reactions to Special Tissues: Certain pulmonary lesions suggest that specific tissues can elicit in some persons a reaction like that to a foreign protein. Thus in a young medical officer who had suffered repeated episodes of pulmonary disease, a lung biopsy revealed bronchial cartilage in process of destruction and absorption, lying within tissue containing a predominance of plasma cells. The lining membranes of the bronchi had in part become ulcerated and replaced by palisaded epithelial cells. Sarcoid-like granulomas were also present in the parenchyma, which in addition showed non-specific changes associated with bronchial obstruction. In this patient there was no arthritis nor other evidence of a generalized reaction to cartilage. It is remarkable also that in a typical case of chondromalacia (63, 64) there were lesions indistinguishable from sarcoid in the bronchial lymph nodes and in the pulmonary parenchyma. The patient was a woman aged 63 who for five years had suffered from arthritis, and gradually lost the cartilaginous portion of her nasal septum, and experienced at first pain and swelling and then shrinkage of the ear cartilages. Similar granulation tissue surrounded many of the bronchial cartilages while others appeared to be in a quiescent state of extreme atrophy.

Apparently analogous reactions to elastic tissue can occur. Some may be simply in response to mineralization of the elastica



Figures 46, 47. Chondromalacia (von Meyenburg-Vehlinger)—auto-immune reaction to cartilage? The reaction in this middle-aged woman was generalized with arthritis, pain and shrinkage of ear lobes, collapse of nasal septum, and death from narrowing of airways five years after onset of symptoms. Atrophy of cartilage with collapse of bronchus, the mucous membrane of which is consequently much folded (Fig. 46). The much-thickened perichondrium and the shrinkage and crowding of the cartilage cells, especially at the periphery, is better shown at higher magnification (Fig. 47). Multiple granulomas in bronchial lymph nodes were indistinguishable from those of sarcoidosis and similar lesions were scattered in the pulmonary parenchyma.

Figures 48, 49. "Giant cell arteritis" of the lung with sarcoid-like reaction—auto-immune reaction to elastic tissue? Especially the outer elastic lamina of the pulmonary artery has become fragmented and there are many giant cells and scattered lymphocytes and plasma cells. Some of the giant cells contain fragments of the elastica. An accumulation of cellular fibrous connective tissue narrows the lumen (Fig. 48). In the parenchyma are scattered non-caseating granulomas (Fig. 49).

as seen in hemosiderosis associated with chronic hemorrhage, and cannot be construed as allergic reactions. Also in sarcoidosis the granulomas may be concentrated about the elastic tissue of blood vessels and bronchi. Lesions resembling giant cell arteritis also occur in the pulmonary vessels. Here the lesions are not typical of sarcoidosis, although additional non-caseating granulomas can occur in the parenchyma. The relationship of the giant cells specifically to the outer layers of the elastica is difficult to explain unless this layer acts as a barrier or as an absorbing surface for substances in transit through the wall.

Goodpasture's Syndrome: The designation "Goodpasture's Syndrome" has been applied, rather incongruously, to the association of repeated pulmonary hemorrhages, anemia, lassitude, fever and loss of weight with progressive glomerulonephritis (65). The relationship to "essential pulmonary hemosiderosis" has not as yet been clarified. Although, occasionally, pulmonary arteriolitis has been demonstrated, the source of the hemorrhage usually is not evident. Elastic reduplication with iron incrustation, especially of venules, must be considered secondary. Ultimately, interstitial fibrosis and hemosiderosis may become severe. The renal lesion is a glomerulonephritis resembling that of Type T of Ellis, but at first involves only individual glomeruli. Death can occur as a result of pulmonary or renal insufficiency or both. A reasonable working hypothesis is that the condition represents an autoimmunity phenomenon to a common antigen in the basement membranes of pulmonary and glomerular capillaries.

Discussion

Of particular interest is the occurrence of lesions resembling those of sarcoidosis in many types of hypersensitivity reactions—in some instances probably preceding the acute episode, as exemplified in the penicillin reaction illustrated in Fig. 4. In the present observations, such lesions have also been found in eosinophilic pneumonia (Fig. 12), angiitis (Fig. 23) including that of the Wegener type (Fig. 27), and in what are probably autoimmune reactions to cartilage. Most significant is the fact that such granulomas are the characteristic response in now well-characterized hypersensitivity reactions of farmer's lung and maple bark disease. Berylliosis, in which similar granulomas are formed, has also been considered an allergic response, since only a small number of the persons exposed become ill. Observations such as these suggest that the sarcoidal granuloma represents one anatomical manifestation of allergy, and that this is at least a rational working hypothesis in considering the pathogenesis of "sarcoidosis".

It is by no means certain that all of the conditions considered in this survey represent antigen-antibody reactions. Some may simply be reactions to abnormal substances deposited in dysproteinemias. What remains to be done is to determine the actual proteins involved and whether hypersensitivity does in fact exist. The mere presence of antibodies does not necessarily indicate the presence of disease, and it is a presumption that the lesions are the consequence of hypersensitivity. In each instance, specific antigens and antibodies must be identified and the distribution of these materials as well as of their complexes in the tissues must be established in relation to the lesions. The actual mechanism of damage to tissue also remains obscure, even when these substances are identifiable in association with the lesions. It must be kept in mind that damage may result not only from direct effects of such complexes but also of the products—for example, vasoactive amines—related to their formation or breakdown or action on other cells or substances. Also the possibility that certain agents may directly release histamines when they are inhaled must be considered, as it has been in byssinosis.

While the precise nature of some of the

lesions described is still moot, it is nevertheless certain that allergic reactions in the lung can have a number of quite diverse microscopic appearances. These can consist of acute exudative reactions with formation of hyaline membranes, exfoliative bronchitis, the hallmarks of long-maintained and repeated bronchospasm and mucous plugging, eosinophilic pneumonias, vasculitis and "granulomatosis", interstitial lymphocytic and plasma cell infiltrations accompanied by non-caseating granulomas, and evidences of reactions to specific components of tissues such as cartilage and possibly elastica and basement membranes suggesting phenomena of autoimmunity.

REFERENCES

1. Osler, W.: On the visceral complications of erythema exudativum multiforme. Am. J. Med. Sci. **110:** 629–646, 1895.
2. Klinge, F.: Die Merkmale der hyperergischen Entzündung. Klin. Wchnschr. **6:** 2265–2267, 1927.
3. Fried, B. M.: Allergic lobar pneumonia; experimental study. J. Exp. Med. **57:** 111–119, 1933.
4. Herbut, P. A., and Kinsey, F. R.: Transitory pulmonary infiltrations (Loeffler's syndrome) in rabbits. Arch. Path. **41:** 489–502, 1946.
5. Cannon, P. R., Walsh, T. E., and Marshall, C. E.: Acute local anaphylactic inflammation of the lungs. Am. J. Path. **17:** 777–784, 1941.
6. Klinger, H.: Grenzformen der Periarteritis nodosa. Frankfurt. Ztsch. f. Path. **42:** 455–480, 1931.
7. Wegener, F.: Über eine eigenartige rhinogene Granulomatose mit besonderer Beteiligung des Arterien-systems und der Nieren. Beitr. z. path. Anat. u. z. allg. Path. **102:** 36–68, 1939.
8. Löffler, W.: Zur Differential-diagnose der Lungeninfiltrierungen. II. Über flüchtige Succedan-infiltrate (mit Eosinophilie). Beitr. zur Klinik Tuberk. **79:** 368–382, 1932.
9. Campbell, J. M.: Acute symtoms following work with hay. Brit. Med. J. **2:** 1143–1144, 1932.
10. Towey, J. W., Sweany, H. C., and Huron, W. H.: Severe bronchial asthma apparently due to fungus spores found in maple bark. J.A.M.A. **99:** 453–459, 1932.
11. Bramwell, J. C., and Ellis, R.: Report of the departmental committee on dust in cardrooms in the cotton industry (Home Office). pp. 36. H. M. Stationery Office, 1932.
12. Léon-Kindberg, M., Adido, P., and Rosenthal, L.: Pneumopathie a éosinophiles. Presse méd. **48:** 277–278, 1940.
13. Frimodt-Möller, C., and Barton, R. M.: A pseudo-tuberculous condition associated with eosinophilia. Indian Med. Gaz. **75:** 607–613, 1940.
14. Weingarten, R. J.: Tropical eosinophilia. Lancet **I:** 103–105, 1943.
15. Cardell, B. S.: Pathological findings in deaths from asthma. Internat. Arch. Allergy & Applied Immunol. **9:** 189–199, 1956.
16. Cardell, B. S., and Pearson, R. S. B.: Death in asthmatics. Thorax **14:** 341–352, 1959.
17. Reeder, W. H., and Goodrich, B. E.: Pulmonary infiltration with eosinophilia (P. I. E. syndrome). Ann. Int. Med. **36:** 1217–1240, 1952.
18. Crofton, J. W., Livingstone, J. L., Oswald, N. C., and Roberts, A. T. M.: Pulmonary eosinophilia. Thorax **7:** 1–35, 1952.
19. Hall, J. W., III, Kozak, M., and Spink, W. W.: Pulmonary infiltrates, pericarditis and eosinophilia: a unique case of the pulmonary infiltration and eosinophilia syndrome. Am. J. Med. **36:** 135–143, 1964.
20. Bell, R. J. M.: Pulmonary infiltration with eosinophils caused by chlorpropamide. Lancet **I:** 1249–1250, 1964.
21. Pepys, J., Riddell, R. W., Citron, K. M., Clayton, Y. M., and Short, E. I.: Clinical and immunologic significance of Aspergillus fumigatus in the sputum. Am. Rev. Resp. Dis. **80:** 167–180, 1959.
22. Maier, C.: Das flüchtige eosinophile Lungeninfiltrat; zusammenfassende Ergebnisse von über 100 Beobachtungen. Helv. Med. Acta **10:** 95–99, 1943.
23. Von Meyenburg, H.: Das eosinophile Lungeninfiltrat; pathologische Anatomie und Pathogenese. Schweiz. med. Wchnschr. **72:** 809–811, 1942.
24. Saupe, E.: Über Lungenröntgenbefunde, insbesondere Infiltrate, bei Asthma. Fortschr. a.d. Gebiete d. Röntgenstr. **61:** 65–84, 1940.
25. Bayley, E. C., Lindberg, D. O. N., and Baggenstoss, A. H.: Loeffler's syndrome; report of a case with pathologic examination of the lungs. Arch. Path. **40:** 376–381, 1945.
26. Liebow, A. A., Steer, A., and Billingsley, J. G.: Desquamative interstitial pneumonia. Am. J. Med. **39:** 369–404, 1965.
27. Moore, R. D., and Schoenberg, M. D.: Alveolar lining cells and pulmonary reticuloendothelial system of the rabbit. Am. J. Path. **45:** 991–1006, 1964.
28. Moore, R. D., and Schoenberg, M. D.: The re-

sponse of the histiocytes and macrophages in the lungs of rabbits injected with Freund's adjuvant. Brit. J. Exp. Path. **45:** 488–498, 1964.

29. Rupp, J. C., Moore, R. D., and Schoenberg, M. D.: Stimulation of the reticuloendothelial system in the rabbit by Freund's adjuvant. Arch. Path. **70:** 43–49, 1960.
30. Liebow, A. A., and Hannum, C. A.: Eosinophilia, ancylostomiasis and strongyloidosis in the South Pacific area. Yale J. Biol. Med. **18:** 381–403, 1946.
31. Carter, H. F., Wedd, G., and D'Abrera, V. St. E.: Occurrence of mites (Acarina) in human sputum and their possible significance. Ind. Med. Gaz. **79:** 163–168, 1944.
32. Christoforides, A. J., and Molnar, W.: Eosinophilic pneumonia. Report of two cases with pulmonary biopsy. J.A.M.A. **173:** 157–161, 1960.
33. Raich, R. A., and Gleason, D. F.: Pulmonary symptoms and eosinophilia due to filariasis. Tubercle **40:** 462–465, 1959.
34. Rose, G. A., and Spencer, H.: Polyarteritis nodosa. Quart. J. Med. **26:** 43–81, 1957.
35. Godman, G. C., and Churg, J.: Wegener's granulomatosis: pathology and review of literature. Arch. Path. **58:** 533–553, 1954.
36. Carrington, C. B., and Liebow, A. A.: Limited forms of angiitis and granulomatosis of Wegener's type. (In press: Am. J. Med.)
37. Churg, J., and Strauss, L.: Allergic granulomatosis, allergic angiitis and periarteritis nodosa. Am. J. Path. **27:** 277–294, 1951.
38. Ehrlich, J. C., and Romanoff, A.: Allergic granuloma of the lung: clinical and anatomic findings in patients with bronchial asthma and eosinophilia. Arch. Int. Med. **87:** 259–268, 1951.
39. Fienberg, R.: Necrotizing granulomatosis and angiitis of the lungs and its relationship to chronic pneumonitis of cholesterol type. Am. J. Path. **29:** 913–931, 1953.
40. Fienberg, R.: Pathergic granulomatosis (Editorial). Am. J. Med. **19:** 829–831, 1955.
41. Kuzma, J. F.: Pulmonary changes in collagen diseases. Dis. Chest. **32:** 365–373, 1957.
42. Bohrod, M. G.: Pathologic manifestations of allergic and related mechanisms in diseases of the lungs. Internat. Arch. Allergy & Applied Immunol. **13:** 39–60, 1958.
43. Aitchison, J. D., and Williams, A. W.: Pulmonary changes in disseminated lupus erythematosus. Ann. Rheumatic Dis. **15:** 26–32, 1956.
44. Doctor, L., and Snider, G. L.: Diffuse interstitial pulmonary fibrosis associated with arthritis. Am. Rev. Resp. Dis. **85:** 413–422, 1962.
45. Meyer, E. C., and Liebow, A. A.: Relationship of interstitial pneumonia, honeycombing and atypical epithelial proliferation to cancer of the lung. Cancer **18:** 322–351, 1965.
46. Caplan, A.: Certain unusual radiological appearances in chest of coal miners suffering from rheumatoid arthritis. Thorax **8:** 29–37, 1953.
47. Caplan, A., Payne, R. B., and Withey, J. L.: A broader concept of Caplan's syndrome related to rheumatoid factors. Thorax **17:** 205–212, 1962.
48. Ramirez-R, J., Lopez-Masano, V., and Schultze, G.: Caplan's syndrome. Am. J. Med. **37:** 643–652, 1964.
49. Gardner, D. L., Duthie, J. J. R., Macleod, J., and Allan, W. S. A.: Pulmonary hypertension in rheumatoid arthritis. Scot. Med. J. **2:** 183–188, 1957.
50. Liebow, A. A.: Pathology of the pulmonary circulation. In: *Arteriosclerosis: A Survey of the Problem.* H. Blumenthal, Ed. Second Edition. C. C Thomas (In press)
51. Rankin, J., Jaeschke, W. H., Collies, Q. C., and Dickie, H. A.: Farmer's lung: physiopathologic features of the acute interstitial granulomatous pneumonitis of agricultural workers. Ann. Intern. Med. **57:** 606–626, 1962.
52. Emanuel, D. A., Wenzel, F. J., Bowerman, C. L., and Lawton, B. R.: Farmer's lung: clinical, pathologic and immunologic study of twenty-five patients. Am. J. Med. **37:** 392–401, 1964.
53. Bishop, J. M., Melnick, S. C., and Raine, J.: Farmer's lung: studies of pulmonary function and aetiology. Quart. J. Med. **32:** 257–278, 1963.
54. Pepys, J., Jenkins, P. A., Festenstein, G. N., Gregory, P. H., Lacey, M. E., and Skinner, F. A.: Farmer's lung: thermophilic actinomycetes as a source of "farmer's lung hay" antigen. Lancet **II:** 607–611, 1963.
55. Emanuel, D. A., Lawton, B. R., and Wenzel, F. J.: Maple-bark disease. Pneumonitis due to Coniosporium corticale. New Engl. J. Med. **266:** 333–337, 1962.
56. Gregory, P. H., and Waller, S.: *Cryptostroma corticale* and sooty bark disease of sycamore (*Acer pseudoplotanus*). Trans. Brit. Mycol. Soc. **34:** 579–597, 1951.
57. Reed, C. E., Sosman, A., and Barbee, R. A.: Pigeon-breeders' lung. J.A.M.A. **193:** 261–265, 1965.
58. Bringhurst, L. S., Byrne, R. N., and Gershon-Cohen, J.: Respiratory disease of mushroom workers: farmer's lung. J.A.M.A. **171:** 15–18, 1959.
59. Prausnitz, C.: Investigation on respiratory dust disease in operatives in the cotton industry.

Great Brit. Med. Res. Counc. Special report series. No. 212, pp. 73. London, H. M. Stationery Office, 1936.

60. Massoud, A., and Taylor, G.: Byssinosis: antibody to cotton antigens in normal subjects and in cotton card-room workers. Lancet **I:** 607–610, 1964.
61. Bouhuys, A., Lindell, S. E., and Lundin, G.: Experimental studies on byssinosis. Brit. Med. J. **1:** 324–326, 1960.
62. Lindskog, G. E., Liebow, A. A., and Glenn, W. W. L.: *Thoracic and Cardiovascular Surgery with Related Pathology*. Appleton-Century-Crofts. New York, 1962.
63. Altherr, F.: Über einem Fall von systematisierter Chondromalacie. Virchows Arch. Path. Anat. **297:** 445–479, 1936.
64. Von Meyenburg, H.: Über Chondromalacie. Schweiz. med. Wchnschr. **66:** 1239–1240, 1936.
65. Rusby, N. L., and Wilson. C.: Lung purpura with nephritis. Annual Rev. Med. **16:** 301–308, 1965.

The National Smallpox Eradication Program: India's Struggle Against Smallpox[1]

By MARSHALL J. KIESELSTEIN, A.B.[2]

DESPITE the fact that man has known how to prevent smallpox since the end of the eighteenth century, the disease remains a major health problem in many parts of the world and is endemic in Africa, Asia and parts of Latin America. In 1963 the World Health Organization reported 99,000 cases of smallpox with a mortality of 25%. India alone reported some 60,000 cases, or nearly two thirds of the world's total. Although smallpox has been essentially eliminated from western Europe and the United States, it is seen occasionally: in 1962 Great Britain had 66 cases while West Germany had 38; in 1963, Poland reported 96 cases and Sweden reported 25 (1). There have been no outbreaks in the United States since 1949 and the last single case was reported in New York City in 1962 (2).

In 1806 Thomas Jefferson wrote to Edward Jenner and prophesied that "future nations will know by history only that the loathsome smallpox has existed and by you has been extirpated" (3). This is undoubtedly true in the western world. In the United States, very few physicians have even seen smallpox and for the most part it is a text-book disease. Yet, in many underdeveloped countries smallpox is still the cause of death, disfigurement and human misery. The pock-marked faces of all too many Indian children are mute evidence to the failure of mankind to defeat this dread disease. It is indeed a sad commentary on medicine that smallpox still exists. As Dr. M. C. Candau, Director General of the World Health Organization, put it: "In relation to our present knowledge smallpox is a true anachronism" (1).

However, great progress has been made in the eradication of smallpox where it still exists. In the past 15 years the incidence of smallpox has been reduced by more than 80%. In 1951 there were approximately 500,000 cases, while in 1958 there were 235,000 and in 1963 only 99,000 (1). Since 1958, five countries participating in the world eradication program have completely eliminated smallpox—Bolivia, Paraguay, Ceylon, Iraq and Lebanon—while four others have completed their eradication programs although they still have some endemic areas—Liberia, Colombia, Cambodia and Iran (4).

Much of the credit for this Herculean feat is due the WHO which has aided many nations in their struggle against smallpox. As an example of the work being done to eliminate the disease, the National Smallpox Eradication Program of India (NSEP) is particularly interesting.

The WHO Smallpox Eradication Program

On the basis of control programs in three different parts of India and on earlier records, WHO experts developed a three-phase program for the eradication of smallpox in endemic areas. The WHO program may be summarized in the following manner (5):

Preparatory Phase: This stage involves a

[1] William Harvey Perkins Prize Essay VII, Section on Public Health, Preventive and Industrial Medicine, The College of Physicians of Philadelphia, 1965. This study was done during the summer of 1965 through a scholarship granted by the Experiment in International Living for travel in India and Kashmir.

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complete epidemiological evalution of smallpox in the target area, the formulation of a definite plan of attack, recruitment and training of personnel, acquisition of sufficient amounts of vaccine and the equipment to properly store and distribute it, and initiation of a public health education program informing the people of the importance of smallpox eradication and the success of the vaccine in achieving this end.

Attack Phase: This is the phase of actual vaccination. It should be concentrated in the most densely populated endemic areas from which the disease is most likely to spread. Only after such areas are completely covered should vaccination begin in adjacent areas. This approach is particularly essential in areas where the vaccine is available in limited quantities, as it is in most endemic areas. Vaccinations and revaccinations should be administered with potent vaccine by properly trained personnel. In order to insure that they "take", all vaccinations and revaccinations should be examined after 6–8 days. Those individuals not showing a major reaction must be revaccinated. Particular attention should be paid to highly susceptible age groups, newborn children and pregnant women, whose mortality rates are very high. The attack phase should be completed within six months.

Control or Maintenance Phase: This stage begins immediately after the attack phase and requires the maintenance of a high level of immunity in a population nearly 100% immunized. This goal must be achieved through vaccination of all newborn children, immigrants and migrant populations. To insure a constant high level of immunity, the entire population should be revaccinated every five years and school age children at the ages of 4, 8, 12 and 16 years (6). Complete epidemiological investigations should also be made of all outbreaks or sporadic cases, and spread prevented by surveillance of those who had contact with the infected patient, by use of anti-vaccinial gamma globulin, chemotherapeutic prophylactics and revaccination.

Smallpox will be considered successfully eliminated from a country only when there has been no indigenous disease for three successive years and when local outbreaks resulting from imported cases are quickly controlled (5).

The National Smallpox Eradication Program

The attack phase of this program was launched in the last quarter of 1962 and by March 1965 it was expected that the goal of 80% vaccination of all sectors of the population would be achieved. At the end of November 1964, 290 million vaccinations had been administered, including 33.75 million primary and 256.25 million revaccinations out of a total population of 439 million. Thus, at that time, approximately 66% of the population had been vaccinated (6).

At the initiation of the eradication program, it was projected that 80% of the population would have to be vaccinated for the success of the program. The initial figure of 80% was based on work in other countries where interruption of transmission had been achieved with a minimum of 80% coverage. However, the evaluation of the program in several areas of India proved the figure 80% invalid for the unique Indian situation. It was found that even in areas where more than 80% of the population had been vaccinated smallpox still occurred in unvaccinated segments of the population (6, 7). As a result of this observation, the goal of the program has been changed from 80% to 100% vaccination. All states will attempt to achieve this goal as soon as possible (6). This extension of the attack phase will be considerably more difficult than the previous vaccination projects. Achievement of the 100% immunization goal may well be impossible

in the near future because of the many problems encountered.

The Problems

Despite the great progress made by the NSEP, many problems still prevent the complete eradication of smallpox from India. These problems fall into two general categories: technical and social.

There can be no doubt that technical problems have long hindered India in her fight against smallpox. In the past, one of the greatest problems had been the maintenance of the potency of smallpox vaccine under tropical conditions. Liquid vaccine deteriorates quickly even at moderate temperatures and after short exposure to sunlight. Vaccine stored for more than seven days at 0°C.–10°C. and for more than 24 hours at higher temperatures should be discarded as its potency will be markedly reduced (5). Thus, Indian vaccination programs were often conducted with vaccine of relatively low potency which gave little or no immunity. The development of freeze-dried vaccines has largely overcome this difficulty. These products are much more stable and will last for months in the tropical climate. They became as labile as the liquid vaccines only after reconstitution (5). Because at the present time India is unable to manufacture sufficient quantities of her own freeze-dried vaccine, the U.S.S.R. has supplied her with some 370 million doses of vaccine and will continue to do so until the entire population has been vaccinated (6). In addition, the WHO is helping to train Indian personnel in the manufacture of freeze-dried vaccine. With the commencement of indigenous production, it is expected that India will soon be able to manufacture enough freeze-dried vaccine to meet the projected requirements of the maintenance phase—120 to 130 million doses per year (6).

Still other technical problems have hindered the NSEP. Evaluation of the program in various areas has repeatedly shown that reported 100% vaccination coverage is not synonymous with complete community protection against smallpox. Besides the loss of potency of vaccine under field conditions, there were many more reasons for this discrepancy. Improper vaccination techniques were used by poorly or hastily trained workers. Incomplete and even dishonest inspection of vaccinated persons was not uncommon, and in many instances evaluating teams were unable to find vaccination scars in "successful" primary vaccinations. Incomplete registration of births often led to incomplete primary vaccinations and to the accumulation of susceptible patients. Revaccination programs which covered mainly cooperative sectors of the population and missed hard core evaders contributed to the noted discrepancy. Finally, incomplete record-keeping and inadequate epidemiological investigation and anti-epidemic measures were also detrimental to the success of the program (8, 9).

However, perhaps the most important obstacles to the success of the NSEP are man's oldest and most pernicious social problems: illiteracy and superstition. Between 1950–51 and 1963–64 India's literacy percentage increased only from 16.6 to 24.0 or about 50% (10). Thus, over 75% of India's 450 million people are illiterate and often fearfully superstitious of anything new and different. In a program like the NSEP where the population is directly involved, public cooperation is absolutely essential if the program is to succeed. However, primitive customs and beliefs often influence the people actively to avoid vaccination and even to hide smallpox victims from the authorities.

As an example of these social problems and how they interfere with the success of NSEP, as well as with all human progress, let us examine the case of the Adivasis community in the 1961 Bihar State Pilot Program (11). Although 89% of the population of the Ranchi district was vaccinated, the

Adivasis refused vaccination on religious grounds. They believed that smallpox is caused by the wrath of the goddess "Sitla Mata" and that the way to prevent, as well as cure, smallpox is by Puja, or prayer meeting. At the Puja, the priest builds a smoky fire and as the house fills with smoke, prayers are recited to drive away the evil. At the end of the prayer recitation a chicken or goat is slaughtered and everyone leaves the Puja site without looking back. In order to protect themselves from infection, people burn sandalwood or ghee (fat) producing a scent which drives away the ghosts. The smallpox patient is worshipped by the flowerman or the most religious person of the family, as it is believed that the goddess resides within the patient. In order to drive smallpox away, anything that remains after worship is put in an earthen pot and thrown out of the village. Shaving, hair-cutting, the household use of ghee and oil, and non-vegetarian diets are prohibited until the final Puja. Also of considerable importance is the restriction that until the final Puja no one is allowed to leave or enter the village. On the fourth or seventh day the patient is given a bath, and a Puja with a chicken sacrifice follows. During this time the patient is fanned and cooled with neem leaves and twigs. But the most noteworthy restriction is that treatment and vaccination are strictly prohibited as they may displease the goddess (11).

Although the preceding description was only of one small group in north central India, such beliefs are common throughout the entire country. In the summer of 1964 I encountered a similar case in South India—over 1000 miles from the one just mentioned. While on rounds with a public health nurse in a little village some 15 miles from the Christian Medical Center at Vellore (90 miles west of Madras), we discovered a smallpox patient in a small Hindu temple on the outskirts of the village. The man was completely covered with pustules and obviously delirious with fever. When we asked some of the village elders why this had not been reported to the health authorities, they answered that since smallpox was a punishment from the gods the best place for the patient was in the temple. They wanted no part of vaccination and insisted that the Pujas they performed would be sufficient. The patient was covered with a green paste which was made of neem leaves. This is of no medicinal value but is a common practice which frequently results in the secondary infection of open pustules.

Before the steps undertaken to overcome this monumental ignorance are discussed, it should be noted that people often refuse vaccination for other than religious reasons. Many farm workers will not allow themselves to be vaccinated for fear they will become ill. Many refuse vaccination simply because as ignorant, illiterate people they fear the new and unknown and do not appreciate the importance of the smallpox eradication program

The great need for public health education in the NSEP became particularly apparent after the 1960–61 pilot project in Uttar Pradesh, India's most populous state. It was found that 46% of the population would accept vaccination with little persuasion, 29% needed a bit more persuasion, and 25% were classified as hard core evaders who would need intensive health education before they would accept vaccination (12).

Accordingly, in order to overcome the reticence of certain segments of the population and to interest sufficiently and persuade the general public to accept vaccination, an intensive program of health education was initiated. In the health education programs which precede mass vaccinations the following facts are presented to the public as simply as possible:

(1) Smallpox is a highly contagious disease which is caused by a virus.

(2) Vaccination and revaccination are the only sure ways to prevent smallpox.

(3) Successful vaccination of 100% of the population can eliminate this disfiguring and often fatal disease as it has in other countries.

(4) If 100% of the population is not vaccinated and sporadic outbreaks are not treated immediately, the susceptible parts of the population will increase and help reintroduce smallpox.

(5) Vaccination reactions occur in those few individuals who have no immunity against smallpox and only indicate that the body is developing resistance to the disease. Therefore, mild reactions following vaccination should not cause alarm.

(6) Smallpox is a dangerous disease which causes blindness, deafness, facial disfigurement, physical disability and death. It not only affects the individual, who is its unfortunate victim, but also injures the community and the nation.

(7) Much money is being spent on the NSEP and it would be wasted if any person is negligent in accepting vaccination for himself or for any members of his family (9, 12, 13).

Of particular interest is the education program used with the Adivasis because it serves as a model for dealing with other groups which evade vaccination. Quite realistically, it was decided that the program should be so designed as to take maximum advantage of those beliefs which were beneficial while diplomatically avoiding controversy over those beliefs which were neither beneficial nor harmful. However, beliefs which were harmful were cautiously and politely discouraged.

Accordingly, local beliefs were divided into three categories:

1. Those which were beneficial for the cure and/or prevention of smallpox.
2. Those which were neutral and had no effect on the cure and/or prevention of smallpox.
3. Those which were harmful.

Thus, isolation of the patients and the prohibition on leaving or entering the village were vigorously encouraged, but no comment was made on the use of neem twigs and other useless customs. To gain more support for the program, the following slogan was popularized: "Worship the goddess but to please her take vaccination" (11). In this slogan can be seen the marriage of the old and the new which has been a characteristic of Indian civilization throughout the ages.

The Progress

The NSEP has been quite successful. As the following figures (6, 7) indicate, there has been a marked reduction in the incidence and mortality of smallpox during the cyclic epidemics:

Period	*Cases*	*Deaths*
1950-1951 (Nov.–May)	224,510	57,305
1957-1958 (Nov.–May)	141,920	37,488
1963-1964 (Nov.–May)	26,077	7,124

Stated another way, for every 100 deaths during the 1950–51 epidemic there were 65 deaths during the 1957–58 epidemic and 13 deaths during the 1963–64 epidemic. Thus, between the first and second epidemics the mortality was reduced 35%, while between the second and third it was reduced 81% (6, 7). This more than twofold reduction in mortality is undoubtedly due to the NSEP.

The Future

We have seen that programs like the NSEP can significantly reduce the incidence of smallpox in endemic areas. Yet, only by dint of conscientious national programs and international cooperation can the disease be eradicated. The final elimination of this medical anachronism will not be easy, but two facts—that man is the only carrier and that vaccination protects—make the complete elimination of smallpox well within the realm of possibility.

REFERENCES

1. Dubourg, Alain-Yves: International Exchange. Abbott-Tempo **3**:(4) 36, 1965.
2. Gordon, John E.: *Control of Communicable Diseases in Man*, 10th ed., American Public Health Association, 1965, p. 215.
3. Hobson, W. *World Health and History*. p. 61, John Wright and Sons, Ltd. Bristol, 1963.
4. W.H.O. Chronicle **17**:8, 1965 from Smallpox Today, Swasth Hind. **7**:(9) 265–268, 1963.
5. W.H.O. Technical Report Series, No. 283, W.H.O. Expert Committee on Smallpox, pp. 17, 24–27, 1965.
6. Ministry of Health Annual Report 1964–65, Government of India, pp. 12–14.
7. Chakravarti, G. P.: Smallpox Eradication Program—Entering the Maintenance Phase. Swasth Hind. **8**:(9) 290–292, 1964.
8. Seal, S. C.: Assessment of Smallpox Eradication Program. Swasth Hind. **7**:(9) 253–259, 1963.
9. Krishnaswami, A. K.: Evaluation of Smallpox Eradication Program and Its Lessons. Swasth Hind. **9**:(3) 58–60, 1965.
10. Indian Pavilion—New York World's Fair 1964–1965, Ministry of Information and Broadcasting.
11. Jamuar, B. P.: Smallpox Eradication in Bihar. Swasth Hind. **7**:(9) 269, 282, 285, 1963.
12. Bharara, S. S.: Health Education in Smallpox Eradication. Swasth Hind. **8**:(2) 45–47, 1964.
13. Cheb, G. W.: Observance of Smallpox Eradication Week—Some Suggestions, Swasth Hind. **7**:(9) 270–281, 1963.

Dr. Henry Wirz and Andersonville Prison: A Matter of Justice[1]

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THE story of Andersonville Prison has been rejuvenated by the publication of McKinley Kantor's novel, "Andersonville", and by the popular play, "The Andersonville Trial". A movie is also soon to be released on this most notorious of Civil War prison camps.

One has only to have modest interest in the medical aspects of the Civil War to encounter the story of Andersonville Prison and the diseases which decimated its inmates. The commandant of such a prison is an intriguing figure, particularly when he is a physician pictured by contemporary authors as a beast and a monster and, in recent historical novels and plays, portrayed as a sadistic psychopath who had much to do with the horror that was Andersonville Prison. The purpose of this paper is to attempt to present this man, Dr. Henry Wirz, and his tragic life as factually as possible and to summarize his "trial" which was, it appears, a travesty of justice.

Andersonville Prison was located at Anderson Station in the lower southwest corner of Georgia, an area of sandy soil and pine trees with a not unpleasant climate. It was built out of necessity because of the steady encroachment of Union forces upon the Richmond-Petersburg area and the attendant difficulties in managing large numbers of enemy prisoners in a crowded city which was experiencing severe difficulties in obtaining supplies.

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The prison was opened on February 27, 1864 under the command of Colonel Alexander Persons, C.S.A., who remained in command until May 1864. The total area was twenty-two acres, housing the sinks, hospital and bake shop, with a fifteen foot high double log wall, the outer wall being a hundred twenty feet from the inner wall. There was a total of 52,345 Union prisoners brought to Andersonville from the time of its opening until it was closed in April 1865. During this time, 13,783 prisoners died and were buried at Andersonville, comprising 26% of the total prisoner population. During a four month period from June 30 to October 30, 1864, there were 10,000 deaths—about one death every twenty-two and a half minutes day and night. The majority of the men died as the result of chronic and acute dysentery combined with malnutrition, scurvy, pyoderma and hospital gangrene.

Although Andersonville Prison was the site of the most deaths in any prison camp of the Union or Confederacy during the war, the percentage of deaths to total prisoners (26%) was matched at the Union camp for Confederate prisoners at Elmira, New York, where, in one winter, 3,000 out of 12,000 Confederate prisoners died of similar causes in the midst of plenty. Notorious northern prison camps also included Camp Chase in Columbus, Ohio, and the disease-ridden Delaware River Island prison camps.

The Life of Henry Wirz

The career of Major Wirz is worthy of scrutiny. If he were the monster that some made him out to be, one would expect to

FIG. 1. Major Henry Wirz, C.S.A. (1822–1865). Reproduced from the collections of the Library of Congress. Courtesy of Dr. John B. Blake, National Library of Medicine.

find evidences of cruelty in his past behavior.

Henry Wirz was born in Zürich, Switzerland, in 1822, of German parents. His family was well-off and he was a graduate of the University of Zürich. He next studied medicine at the University of Paris and the University of Berlin. He subsequently married and fathered two children. His wife having died in 1849, in 1852 he left the children with his wife's parents and came to the United States. During his first year in this country he worked in a woolen mill at Lawrence, Massachusetts in order to learn English, and then went to Louisville, Kentucky, in 1853. He practiced medicine in this community and in 1854 married Elizabeth Wolfe, a widow with two children. In 1855 a daughter resulted from this union. In 1858 the family moved to Madison, Louisiana, where the doctor practiced medicine until the outbreak of the Civil War. He was a successful practitioner, highly thought of in his community and a strong partisan of the South and of States Rights.

His war record was an unblemished one. In June 1861 he joined the Madison Infantry of the Louisiana Volunteers as a private, which was not unusual for a physician in those days. In August, his company moved to Richmond where he was made a clerk in the military prison. In November 1861, while in Tuscaloosa, Alabama, he was promoted to sergeant. In May 1862 he received a major injury to the right forearm, with a compound fracture of the radius, at the Battle of Seven Pines. In the following month he was promoted to Captain and commended for gallantry in action. In August 1862 he was made inspecting officer of prisons in the Richmond district. In November 1862 he was furloughed for medical attention, and in December of that year went to Europe as plenipotentiary, with the rank of major, for President Jefferson Davis and the Confederate commissioners who were seeking aid from France and England. One of the reasons for this assignment was his ability to speak several languages. A French surgeon operated upon his battle-wound osteomyelitic radius in 1863. Major Wirz did not return to the United States for over a year—until February 1864. In April he was placed in charge of the interior of Andersonville Prison and inherited all of its problems and all of its horrors. In May 1865 he was arrested and his trial was held from August 23 until October 16 of that year. He was sentenced to death and hanged in the

courtyard of the Capitol Prison in Washington, D. C., on November 10, 1865 and buried in the prison yard next to Mrs. Azerodt who was hanged for her supposed complicity in the plot to kill President Abraham Lincoln. At the present time Wirz's remains rest in the Catholic Cemetery at Washington, D. C.

Personnel of Andersonville Prison

From February until May 1864, Andersonville Prison was under the command of Colonel Alexander Persons. In June 1864, General John H. Winder was appointed Commandant of the prison and he remained in this position until October 1864, when he died of heart disease. General Winder, a physician, was 70 years of age when appointed Commandant. He had formerly been in charge of war prisons in the Richmond area. When he left Richmond, the *Richmond Times Examiner* editorially stated, "Thank God Richmond is at last rid of old Winder! God have mercy on those to whom he has been sent". General Winder hated Union soldiers. He lived several miles from the prison and, hard as it is to believe, apparently never visited the prison itself during his tenure as commander. He told Colonel Chandler, who was inspector of Confederate prisons, in August 1864, "It was better to see one half of the prisoners die than to take care of them". Chandler's report to President Davis and the Confederate Commissioners, a scathing denunciation of Winder, recommended his immediate replacement. It is interesting to note that no mention was made at the time of Henry Wirz.

W. Sidney Winder, son of General Winder, was the Adjutant of the Georgia post. It was his responsibility to execute the details and orders which applied to the troops on duty. He was also in charge of the records and in command of the Provost Marshal. Therefore he was actually second in command to General Winder. Richard B. Winder, who was General Winder's nephew, was the quartermaster. He handled all the supplies for troops and prisoners and was responsible for food distribution. Major Wirz was actually fourth in command of the prison and thus responsible solely for its interior condition.

Efforts of the Confederates and of Henry Wirz to Improve Andersonville Prison

Early in the Civil War, the Union Command decreed that all medical supplies were contraband. This meant that only a trickle entered the Confederacy by means of blockade runners and, as a consequence, the people of the Confederacy, its troops and its prisoners of war suffered alike for lack of medical supplies.

Major Wirz, when first placed in charge of the prison, had all drummer boys and other youths attached to the Union troops moved out of the prison itself. They lived in a separate camp under more satisfactory conditions. In June 1864, Henry Wirz allowed the trial of the prisoner gangs who were preying on other prisoners, which resulted in six gangsters being hanged. He was also responsible for enlargement of the stockade by some nine acres and the placing of a hospital, such as it was, outside of the stockade. James M. Page, a Union prisoner who wrote a book entitled, "The Truth About Andersonville", recalled Wirz with a kindly and understanding pen. At one time, when Page was discussing with Wirz the problems of the prisoners, the former stated that Wirz looked at him with tears in his eyes and said, "God help you; I cannot". In June 1864, Wirz wrote to Sidney Winder concerning the poor commissary and requested better food and supplies. In August of that year he wrote Colonel Chandler, the Inspector General of Confederate Prisoners, asking for tools so that the housing shelters could be improved. Wirz made daily rounds in the prison, without arms, and was never molested—a strange degree of immunity for one as much

hated by those who would later write about him.

In July 1864, the Confederacy allowed four prisoners to go to Washington to plead for prisoner exchange. These men went on their honor and subsequently returned when their pleas were unsuccessful.

The eight physicians on the staff at Andersonville repeatedly implored General Winder to improve conditions. Southern civilians who brought food and vegetables to the prison were refused admission by Winder's order.

It is important to realize that the Confederate military and political leaders knew of Andersonville and its horrors. They were involved in a war which they knew they were losing and were fighting the final battles in desperation. Under such stress, they had relatively little thought about the problems of military prisons.

Efforts of the Confederacy to Exchange Prisoners

In August 1864, Union Secretary of War Benjamin Stanton stated, "We do not propose to reinforce the rebel army by exchanging prisoners. We will not exchange able-bodied men for skeletons". In the same month, General Ulysses S. Grant wrote to Mr. Stanton, "Under no circumstances will there be any authorization for prisoner exchange".

Colonel Ould, the Confederate Commissioner of Exchange, in August 1864 offered to give up all sick and wounded Union prisoners without requiring equivalent exchange. Nothing was done about exchange of these sick and wounded prisoners, however, until December 1864—after the summer had had a chance to reap its grisly toll in the prison camps of both the North and the South.

James Page stated in his book, "The Truth About Andersonville", that the prisoners hated Stanton far worse than any of the prisonkeepers because of his denial of exchange.

It is also a matter of record that civilian groups and the U. S. Sanitary Commission in the North requested permission to take food and supplies to Andersonville Prison. This mercy mission was refused by Secretary of War Stanton.

The leadership of the Union realized that prisoners held by the Confederates required food, shelter, and guards, and so indirectly contributed to the Northern war effort. Andersonville was no secret to the Northern leadership. It was a price they were willing to pay in order to win the war in the shortest possible time.

Dr. Joseph Jones in Andersonville

Dr. Joseph Jones, an 1856 graduate of the University of Pennsylvania Medical School, was a roving Medical Inspector for the Confederacy. He was at Andersonville Prison during August and September 1864, staying there for several weeks. He sent a detailed report to the Confederate Surgeon-General concerning Andersonville. (Prior to the outbreak of the war, Dr. Jones was professor of chemistry in the Medical Department at the University of Louisiana in New Orleans. He was also the author of a 300-page book on hospital gangrene, published by the U. S. Sanitary Commission in 1871. Following the war, Dr. Jones joined the faculty at the University of Georgia and his academic career was a distinguished one.) His report on Andersonville Prison noted:

1. 'Sweet Water Creek', a source of water for prisoners, was contaminated by human excrement and other filth. It contained maggots, animaliculae and cryptogenous plants.

2. Fleas flourished in the sandy soil and mosquitoes swarmed in great numbers.

3. There were 9,479 deaths (30%) among the entire number of prisoners from February to September 1864.

4. A horrible stench arose from the combination of human dung and filth covering the low grounds of the camp. The dis-

eases most prevalent were acute diarrhea and chronic dysentery.

5. Human skeletons cried out for food and medical attention in the prison hospital; flies swarmed down their open mouths and lice crawled over their clothes. Men brought into the hospital from the stockade were so begrimed from head to toe in their excrement and so black from smoke and filth that they resembled Negroes rather than white men in appearance. Men preferred to die by slow starvation rather then eat the husk-corn meal and die an agonizing death from lacerated intestines.

Dr. Jones' report in September of 1864, describing Andersonville as a "great mass of misery", was sent to Confederate Commissioner Alexander Stevens who in turn forwarded it to Jefferson Davis. Nothing was done.

The Trial of Henry Wirz

When General Joseph E. Johnston surrendered to General William T. Sherman, an amnesty was granted to all Confederate soldiers. Despite this fact, following the surrender, Henry Wirz was arrested and taken to Washington for trial.

The original charges were against President Davis, Confederate Commissioners Seedon and Cobb, J. H., R. B. and W. S. Winder, and several other physicians on the staff at Andersonville. On the second day of the trial, Colonel Norton Parker Chipman, the Prosecutor, later recalled, "I was directed to prepare new charges leaving out Davis, Seedon and others of Davis' cabinet and to proceed against Wirz". The members of the Board at the court martial consisted of six generals, one colonel and one lieutenant colonel. General Lou Wallace, of *Ben Hur* fame, was one of these officers.

The attorneys for Henry Wirz originally were ex-judge Hughes, ex-governors Denver of California and Charles J. Peck of Illinois, and Louis Schade of Washington, D. C., an attorney of Swiss origin. All but Schade withdrew as Wirz's attorneys on the second day, when it was announced that charges had been dropped against all others with the exception of Wirz.

Although there were more than thirteen direct specifications of causes of death of Union prisoners against Henry Wirz, all the victims names were unknown! Three of those he was accused of killing died while he was away on furlough. His absence at the time was documented by Confederate records.

The principal witness against Wirz, Felix Delabaume, was represented as being a grand nephew of General Lafayette. He was a charming, articulate young man who wound a tale of sadistic cruelty and murder about the defendant that greatly influenced all who heard him. He received a commendation from the Trial Commission for his efforts and was rewarded with a position as a clerk in the Department of Interior. Eleven days after Henry Wirz's hanging, however, some Washington, D. C. citizens of German descent recognized him as Felix Oeser, a Union Army deserter from the New York Seventh Regiment. Secretary Harland abruptly dismissed him from his politically-appointed clerkship, but he received no other punishment.

It is interesting that Colonel Chipman's book, which he wrote in 1911 in defense of the conduct of the Andersonville trial, did not mention the events subsequent to this man's testimony—events which proved Oeser to be a fraud and a liar. Chipman described him only as the star witness for the prosecution. His book is of interest for it mirrors the guilt that its author felt for his role as prosecutor over a period of forty-five years following the trial.

Among other things, Colonel Robert Ould, who was the Confederate Commissioner of Exchange, was subpoenaed to testify in Henry Wirz's behalf, but was threatened with arrest if he came to Washington. Father Boyle, a Roman Catholic priest, was Wirz's spiritual advisor and a

man of unquestionable integrity. On the day prior to Henry Wirz's hanging, unknown emissaries came to his lawyer Louis Schade, Father Boyle and Wirz himself. They stated that if Wirz incriminated Jefferson Davis and others to the extent that they had ordered the starvation and murder of the Andersonville prisoners, then Wirz would be given a prison sentence rather than hanging. Wirz maintained his innocence, his composure and his dignity. He thanked the officers in charge for their treatment of him. One of them described him as "the picture of courage". He was duly hanged and his corpse buried in the yard of old Capitol Prison next to those of the conspirators in Lincoln's death. His wife was refused his body at the time, although in later years it was transferred to the Catholic Cemetery in Washington, D. C.

Conclusion

Dr. Henry Wirz was not a noble figure; neither was he the monster that he was portrayed to be. He was an irascible, chronically-ill man who was saddled with the worst possible assignment in the Confederate Army, under a sadistic superior who hated the Union and all that it stood for. Wirz did not create Andersonville prison and he was not wholly responsible for the horror that it became. The inherent evil and immorality of every war has spawned in one way or another its own Andersonville.

References

1. Tyler, Lyon G.: Major Henry Wirz, The William and Mary Quarterly **27:** 145–152, 1919.
2. Jackson, Daniel: Doctors Afield: Henry Wirz (1822–1865). New England Journal of Medicine **262:** 31–33, Jan. 7, 1960.
3. Elarton, John W.: *Andersonville and National Cemetery,* Burr Publishing Co., Aurora, Neb., 1913.
4. *Harper's Weekly* (New York City), Vol. IX (1865): No. 451, p. 515, Aug. 19; No. 454, p. 562, Sept. 9; No. 455, p. 582, Sept. 16; No. 465, pp. 738, 741, Nov. 25.
5. Anon.: *The Demon of Andersonville,* Barclay & Co., Philadelphia, 1865.
6. Spencer, Ambrose: *Andersonville Prison.* Harper and Bros., New York, 1866.
7. Page, James M., and Haley, M. J.: *The True Story of Andersonville Prison: A Defense of Major Henry Wirz,* Neale Publishing Co., New York, 1908.
8. University of Pennsylvania Medical Alumni in the Civil War. Medical Affairs, Vol. II, Nos. 1 & 2, Spring 1961.
9. Stevenson, R. Randolph: *The Southern Side, or Andersonville Prison,* Turnbull Bros., Baltimore, 1876.
10. *The Trial of Henry Wirz,* Government Printing Office, Washington, D. C., 1868.
11. Kantor, McKinley: *Andersonville,* The World Publishing Co., Cleveland and New York, 1955.
12. McElroy, John: *This Was Andersonville,* Edited by John Meredith, New York, 1957.
13. Chipman, Norton Parker: *The Tragedy of Andersonville,* Blair-Murdock Printing Co., San Francisco, 1911.
14. Barton, Clara: *Report of an Expedition to Andersonville, Georgia, July 1865,* reprinted *in toto* in reference 13.

Ophthalmology at the University of Pennsylvania: A Chronology[1]

By DANIEL M. ALBERT, M.D.[2] AND HAROLD G. SCHEIE, M.D.[3]

1765 Medical School of the University of Pennsylvania founded.

Dr. William Shippen, Jr., Professor of Surgery, Anatomy and Obstetrics at the Medical School of the University of Pennsylvania recognized for skill in eye surgery.

1785 Benjamin Franklin describes bifocals.

1795 Dr. Philip Syng Physick performs cataract extraction using method of Baron Wenzel; possibly first surgeon in America to *extract* cataract.

1797 Dr. Samuel Cooper directs attention of American physicians to the use of mydriatics in his inaugural dissertation to the Medical Faculty of the University of Pennsylvania on effects of *Datura stramonium,* the common jimson weed.

1800 Dr. Physick uses punch forceps of his own design for "artificial pupil".

On Cataract by Frederic Seip, Class of 1800, earliest graduation dissertation from the Medical School of the University of Pennsylvania to deal with ophthalmology.

1801 Dr. John Redman Coxe, Professor of Chemistry in the Medical Faculty is the first Philadelphia doctor to adopt vaccination for smallpox (a disease still responsible for 20% of blindness in India).

1805 Dr. Physick appointed Professor of Surgery; lectures on diseases and surgery of the eye.

1811 Dr. William Gibson perfects his technique for linear extraction of the lens.

1813 *Elements of Surgery* by Dr. John Syng Dorsey published; probably first American surgical text to contain section on ophthalmology.

1817 Elisha North, student of Drs. Benjamin Rush and William Shippen, Jr., opens America's first ophthalmic hospital in New London, Connecticut.

1818 Dr. William Gibson divides rectus muscles for treatment of squint in several patients, but is dissuaded by Physick from further use of the operation.

1819 Dr. William Gibson succeeds Physick as Professor of Surgery; accomplished ophthalmic surgeon and teacher.

Dr. William Gibson describes operation for "absorption of cataract" in which silk thread is passed through lens.

1820 Dr. Isaac Hays, graduate of the Medical School of the University of Pennsylvania, becomes editor of *Philadelphia Journal of the Medical and Physical Sciences* (later *The American Journal of the Medical Sciences*), which at quarterly intervals includes a section reviewing advances in ophthalmology.

1821 Dr. William Gibson devises special type of scissors for dissolution of cataractous lens.

Dr. George McClellan, following graduation from the Medical School of the University of Pennsylvania, opens the Institution for Diseases of the Eye and Ear in Philadelphia—the earliest

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eye hospital in Philadelphia and the third eye hospital in America.

1822 Pennsylvania Infirmary for Diseases of the Eye and Ear, Philadelphia's second ophthalmic institution, opened. Drs. Physick and Gibson appointed consulting surgeons.

1823 First American textbook on the eye is published: *A Treatise on the Diseases of the Eye* by Dr. George Frick of Baltimore, dedicated to Frick's old professor, Dr. Philip Syng Physick.

1823–1826 Dr. Isaac Hays employed to lecture on ophthalmology in Dr. John D. Godman's School of Anatomy.

1824 Dr. William Gibson's *The Institutes and Practice of Surgery* published; contains detailed discussion of diseases of the eye and their treatment.

Dr. William E. Horner, Adjunct Professor of Anatomy, describes "a small muscle of the inner commissure of the eyelid", later known as "Horner's muscle".

1825 Pennsylvania Infirmary for Diseases of the Eye and Ear visited by Dr. Daniel Drake, who establishes Cincinnati Eye Infirmary in 1827.

1828 John McAllister, optician and graduate of the University of Pennsylvania, grinds first astigmatic correction in America.

1834 The Wills Hospital for the Relief of the Indigent Blind and Lame opens its doors, staffed by graduates from the Medical School of the University of Pennsylvania. Drs. Hays, Parrish, Littell and Fox first Attending Surgeons.

First American edition of Sir William Lawrence's *Diseases of the Eye* published, edited by Dr. Isaac Hays.

1837 Dr. Squier Littell's *A Manual of Diseases of the Eye* published.

Dr. William E. Horner describes method of correcting ectropion of the lower lid; probably first plastic ophthalmic procedure to be devised by an American surgeon.

1839 Dr. Isaac Parrish initiates instruction in ophthalmology at the Wills Eye Hospital with a series of lectures on ophthalmic surgery.

1840 Dr. William E. Horner devises operation for treatment of obstruction of lacrimal sac by drilling an opening directly through the lacrimal bone.

1842 Dr. Crawford W. Long of Jefferson, Georgia, a graduate of the University of Pennsylvania, uses sulfuric ether for anesthesia in three operations.

1849 Dr. Isaac Hays' American edition of T. Wharton Jones' *Principles and Practice of Ophthalmic Medicine and Surgery* published.

1853 Dr. John H. Brinton, returning from Paris, introduces first ophthalmoscope to Philadelphia.

1853–60 Dr. Silas Weir Mitchell, in a one-room laboratory, studies cataract formation following administration of sugar to frogs. This work marked the start of experimental ophthalmology in America.

1855 Philadelphia General Hospital establishes ophthalmology wards supervised by general surgeons. Wards used for instruction of medical students.

1864 American Ophthalmological Society founded. Four professors from the Medical School of the University of Pennsylvania subsequently serve as president.

Drs. Isaac Hays, Ezra Dyer, William Hunt and Thomas G. Morton elected to membership in the newly formed American Ophthalmological Society.

Dr. David Hays Agnew, Assistant Professor of Clinical Surgery at the Medical School of the University of Pennsylvania, elected Attending Surgeon to the Wills Eye Hospital.

1870 Drs. William F. Norris and George C. Strawbridge elected instructors in the treatment of eye disease. Both

had recently returned from Europe and the clinics of von Graefe, Arlt, Jaeger and Bowman.

(Oct. 6) Eye Dispensary of the University of Pennsylvania (Ninth and Chestnut Streets) opens its doors. Elective lectures in ophthalmology for medical students begin.

Dr. Norris utilizes wet-plate process for clinical and histologic photographs of the eye.

Asepsis introduced in ophthalmic surgery at the Hospital of the University of Pennsylvania.

Eye dispensary opened at Pennsylvania Hospital.

Ophthalmological Society of Philadelphia founded, forerunner of Section on Ophthalmology of The College of Physicians of Philadelphia. Founders include Drs. Agnew, Norris and Strawbridge. Dissolved in 1875.

1872 Eye dispensary opened at Presbyterian Hospital.

Eye dispensary opened at Children's Hospital of Philadelphia with Dr. George C. Harlan as first ophthalmic surgeon. Dr. Samuel Doty Risley, assistant, provides first correction for mixed astigmatism in Philadelphia.

1873 Dr. Strawbridge pioneers in use of von Hippel trephine in treatment of glaucoma, retinal detachment and transplantation of animal cornea.

1874 Dr. Norris elected first Clinical Professor of Diseases of the Eye, the fourth such chair in the United States. (Earlier chairs were Miami Medical College in Cincinnati, 1860; Bellevue Medical College, 1868; Harvard Medical School, 1871.)

Ophthalmic pathology laboratory established adjacent to eye clinic at University of Pennsylvania.

1875 Dr. Risley publishes *Comparative Value of Mydriatics,* which helps place use of cycloplegics on a firm basis.

1876 Dr. S. Weir Mitchell, Philadelphia neurologist, introduces term "eye strain" into literature, attributing many nervous symptoms to errors of refraction.

1877 "Bedside teaching" of ophthalmology undertaken in medical curriculum, utilizing wards of new University Hospital and Philadelphia General Hospital. Course is elective.

Philadelphia General Hospital appoints first ophthalmologist, Dr. Edward O. Shakespeare.

1878 Section on Ophthalmology of the American Medical Association founded.

1879 Ophthalmology becomes a regular part of medical school course, with lectures given during all three years. By 1880, 120 hours of lectures and demonstration in ophthalmology given. Final examination initiated 1888. In 1892, Norris and Oliver's *Textbook of Ophthalmology* is made required reading.

1883 Third volume of Agnew's *The Principles and Practice of Surgery* published, including 145-page section on the eye.

First post-graduate instruction in ophthalmology offered at the University of Pennsylvania.

The Philadelphia Polyclinic and College for Graduates in Medicine, forerunner of the Graduate School of Medicine of the University of Pennsylvania, established.

1885 Dr. Edward Oram Shakespeare is among the first in Philadelphia to employ bacteriological techniques in diagnosis of conjunctival inflammation. Inventor of popular ophthalmoscope.

Dr. Edward Jackson's *Skiascopy* published.

Dr. George E. de Schweinitz accepts position as Dr. Norris' assistant and begins the study of ophthalmology.

1886 Dr. Strawbridge reports favorably on

topical cocaine for ophthalmic anesthesia, introduced two years earlier by Carl Koller.

1890 Section on Ophthalmology of the College of Physicians of Philadelphia founded. University of Pennsylvania represented by Drs. Norris (Chairman), Randall, Risley and de Schweinitz. Thirteen chairmen since that time have been from the Department of Ophthalmology of the University of Pennsylvania.

Dr. de Schweinitz the first to produce toxic changes in the eye experimentally with quinine.

1891 Title of chair of ophthalmology changed to Professor of Ophthalmology.

Dr. George A. Piersol, once a member of eye dispensary staff, elected to Chair of Anatomy. His books, *Normal Histology* (1893) and *Human Anatomy* (1907), include important sections on the eye.

1892 At Dr. William Osler's urging, Dr. de Schweinitz completes first edition of *Diseases of the Eye,* the leading American ophthalmology text for the next quarter century.

Drs. Norris and Oliver's *Textbook of Ophthalmology* published.

1894 Dr. de Schweinitz receives Alvarenga Prize from College of Physicians of Philadelphia for monograph on the toxic amblyopias.

1896 Drs. Risley and Burton Alexander Randall examine eyes of 1200 Philadelphia schoolchildren, stress importance of school visual examinations, good illumination and properly designed desks.

American Academy of Ophthalmology and Otolaryngology organized. Members of Department of Ophthalmology of the University of Pennsylvania play important role in subsequent direction and development.

First of four volumes of Norris and Oliver's encyclopedic *System of Diseases of the Eye* published.

1897 Dr. de Schweinitz among the first to remove a foreign body, which had been shown in X-ray, from the eye.

Male and female eye wards first opened in new Agnew wing of the Hospital of the University of Pennsylvania.

1899 Dr. Risley describes rotary prism, now known as "Risley prism".

1902 Laboratory of ophthalmic pathology enlarged and re-equipped. Drs. Shumway, Saxon, Baer, Hosmer, How, and DeLong work in laboratory while de Schweinitz is chairman.

Dr. George E. de Schweinitz appointed to the Chair of Ophthalmology at the University of Pennsylvania. Dr. Archibald G. Thomason, expert on industrial injuries and colorblindness, member of ophthalmology staff at the University of Pennsylvania, serves as Ophthalmic Surgeon to Pennsylvania Railroad.

1902–1924 Over forty physicians take preceptorship under de Schweinitz. Twelve of de Schweinitz's pupils become professors of ophthalmology at various American medical schools.

1903 Drs. de Schweinitz and Edsall show metabolic disturbances in patients with tobacco amblyopia.

1903–1904 Eye clinic enlarged and re-equipped by Dr. de Schweinitz. Eye ward capacity, 14.

1904 Drs. de Schweinitz and Shumway describe conjunctivitis nodosa caused by the hairs of the caterpillar *Spilisoma virinica.*

1904–1905 Pennsylvania football team is undefeated. Head coach, Dr. William Sheldon, is Assistant Surgeon in the Dispensary for Diseases of the Eye.

1905 Dr. Edward Shumway reports first microscopic examination of eyes of patient with amaurotic family idiocy.

1906 Dr. de Schweinitz gives first descrip-

tion of tortuosity of arterioles in the macular region in patients with arteriosclerosis.

1907 Dr. Edward Jackson describes crossed cylinder.

Separate eye operating room established in Agnew Pavilion opposite the eye ward.

1907–1919 Expanded post-graduate program offered in ophthalmology at the University of Pennsylvania by Drs. Carpenter, Mellor, de Schweinitz and Shumway.

1910 Infiltration of lid areas with procaine added to cocaine drops in eye surgery.

Dr. Edward Shumway demonstrates that acute contagious conjunctivitis in Pennsylvania area is due mainly to pneumococcus. Also describes corneal complications occurring with Koch-Weeks conjunctivitis.

1913 Dr. Charles A. Oliver establishes prize "to be given to that student in the graduating class in the Department of Medicine who shall annually receive the highest average and pass the best examination in ophthalmology".

1915–1917 Dr. Alan C. Woods, a Research Fellow at the University, begins clinical and experimental work on Elschig's allergic theory and begins development of intradermal test to detect hypersensitivity to uveal pigment and pigment desensitization.

1916 Graduate School of Medicine of the University of Pennsylvania organized. Dr. Thomas B. Holloway formulates curriculum for Department of Ophthalmology.

1917 Dr. Holloway reports studies of pathology of the vitreous. Gives the name "snowball opacities" to asteroid hyalitis.

American Board of Ophthalmic Examination formed with Dr. Edward Jackson first chairman, the first such board in American medicine.

Drs. de Schweinitz and A. C. Woods report on experimental studies of trypanosome keratitis.

1919 Drs. de Schweinitz and Meyer Wiener describe a patient with Cysticercus of the vitreous.

1920–1924 Dr. Alfred Cowan, active in field of optics, devises new axis finder, perimetric chart for measuring retinal lesions and a method for luminous working of ophthalmologic instruments.

1923 Dr. Jonas S. Friedenwald, later Professor of Ophthalmology at Johns Hopkins Hospital, takes preceptorship under Dr. de Schweinitz.

Dr. de Schweinitz the first American to deliver Bowman Lecture in London before Ophthalmology Society of United Kingdom, entitled "Ocular Aspects, Especially Field Defects, of Pituitary Body Disorders".

1924 Slit lamp microscope installed in eye dispensary.

Dr. Thomas B. Holloway appointed to the Chair of Ophthalmology.

Laboratory of perimetry with full-time technician established.

1925 Dr. William T. Shoemaker appointed Chairman of Ophthalmology in the Graduate School of Medicine; serves until 1942.

1928 *Introductory Course in Ophthalmic Optics* by Dr. Alfred Cowan published.

1929–1958 Dr. Wilfred E. Fry, while a member of Department, contributes extensively to field of ophthalmic pathology; edits *Transactions of American Ophthalmological Society* (1942–1959).

1936 Dr. Alexander G. Fewell made temporary chairman of the Department of Ophthalmology, following the death of Dr. Holloway.

1937 Dr. Francis Heed Adler appointed Chairman of the Department; clinics and wards enlarged and rebuilt; resi-

dency program started at the University of Pennsylvania.

1938 *Gifford's Textbook of Ophthalmology* by Dr. F. H. Adler first published. Now in seventh edition.
Ophthalmoscope made required equipment for medical students.

1938–1946 Dr. P. Robb MacDonald associated with Department; contributions include studies of role of melanophore hormone in pigmentary degeneration of the retina, macular sparing following bilateral thrombosis of posterior calcarine arteries and ocular manifestations of vitamin deficiencies.

1940 Dr. H. G. Scheie reports on site of disturbance in Adie's syndrome.

1940–1953 Dr. Irving H. Leopold, while member of the staff at the University of Pennsylvania, makes important contributions to the pharmacology and toxicology of the eye, including the development of DFP in the treatment of glaucoma.

1942 Dr. Edmund B. Spaeth appointed Chairman of Ophthalmology in the Graduate School of Medicine; serves until 1954.

1945 Title of Chair of Ophthalmology changed to William F. Norris and George E. de Schweinitz Professor of Ophthalmology, with funds for endowment provided from estate of Dr. de Schweinitz.

1946 Dr. Scheie describes clinical and histologic ocular changes in scrub typhus.

1949 Dr. Scheie appointed ophthalmologist to the Children's Hospital of Philadelphia.

1950 Dr. Adler appointed Editor-in-Chief, *A.M.A. Archives of Ophthalmology*.
First edition of Adler's *Physiology of the Eye: Clinical Application* is published.

1950–1960 Contributions from the Department of Ophthalmology include articles on gonioscopy; goniotomy and goniopuncture in the treatment of congenital glaucoma; the value of ACTH in the treatment of Herpes zoster and other eye diseases; anesthetic agents and their administration in ophthalmic surgery; the management of ocular trauma; new corneal scissors for cataract extraction and peripheral iridectomy with scleral cautery. Texts from the Department include Adler's *Physiology of the Eye* (1950) and Wiener and Scheie's *Surgery of the Eye* (1952).

1952 Philadelphia Veterans Administration Hospital erected. Ophthalmology Department staffed by faculty and residents of the University of Pennsylvania.

1955 Dr. Irving H. Leopold appointed Chairman of Ophthalmology in the Graduate School of Medicine; serves until 1964.

1960 Dr. Harold G. Scheie appointed to Chair of Ophthalmology. Clinical facilities rebuilt, with support of Margaret Grundy Memorial Fund. Elective five-year training program originated, which permits two years of research and advanced study in related basic sciences.

1960–1965 Contributions of Department to ophthalmic literature include modified technique for aspiration of congenital or soft cataracts, glaucoma surgery, treatment of hyphema with fibrinolysin, description of *forme fruste* of Hurler's disease, histochemical study of the adrenergic and cholinergic innervation of the anterior segment.

1964 Ophthalmology Section of the University's Graduate School of Medicine merged with Medical School Department. Curriculum directed toward a basic science introduction to ophthalmology.

A Silver Pitcher for Dr. Nathaniel Chapman[1]

By ELLA N. WADE[2]

FREQUENTLY gifts are offered for the collections of the College or Mütter Museum, sight unseen, and this occurred recently when Mr. Sydney Thayer, Jr., of Bryn Mawr, called on the telephone to enquire if the College would be interested in accepting a silver pitcher that had been given to Dr. Nathaniel Chapman, his great-great grandfather, by the City of Philadelphia.

While this seemed a gift that would be an interesting and historical addition to the College collections, and acceptable to the Committee on College Collections and the Council, we could not give Mr. Thayer an answer until those responsible for its acceptance had seen it. Dr. Chapman, first president of the American Medical Association, was an eminent Philadelphia physician, yet the only items in the College collection associated with him are his oil portrait by Sully and a silver-headed cane that he carried. This cane has done considerable traveling because of its famous owner and has been on exhibit somewhere almost continuously for the past two years. It was recently on exhibit at the William Penn Memorial Museum and Archives Building in Harrisburg where it remained until August 31, 1966.

Mr. Thayer kindly brought the pitcher to the College, carefully wrapped in tissue and tied with white satin ribbon. It exceeded our expectations. It is of a very graceful design, having a footed base. It is about twelve inches tall and the diameter of the bowl at the widest part is seven inches. A quarter-inch band depicting bunches of grapes and leaves encircles the lowest part of the base, and the same design surrounds the upper part of the base where it meets the vessel itself. A three-quarter inch duplicate of this band decorates the upper part of the bowl. The handle is ornate.

On the front of the pitcher the following is engraved:

To
Dr. Nathaniel Chapman
The City of Philadelphia
Grateful
for his disinterested and
intrepid exertions
in a
Period of Public Calamity
Transeat exemplum

FIG. 1. Silver pitcher presented to Dr. Nathaniel Chapman, March 1833. Mütter Museum, The College of Physicians of Philadelphia.

[1] Prepared and published at the request of the Council of The College of Physicians of Philadelphia.

[2] Curator of the Mütter Museum and Custodian of the College Collections.

The absence of a date was unusual, and Mr. Thayer had no idea when or for what specific event the pitcher was given.

Mr. W. B. McDaniel, Curator of the Library Historical Collections of the College, solved this problem, for in Volume 2 of *Philadelphia; a history of the city and its people,* by E. P. Oberholtzer, he found the following: "Silver pitchers were presented in March, 1833, to thirteen physicians who had taken conspicuous parts in treating the sick as chief officers of the various hospitals. These were as follows: John K. Mitchell, Nathaniel Chapman, Joseph Parrish, John C. Otto, Thomas Harris, Samuel Jackson, Charles Lukens, William E. Horner, Charles D. Meigs, Hugh L. Hodge, Richard Harlan, Oliver H. Taylor, and G. Emerson." Here it was also learned that the epidemic indicated was Asiatic cholera in 1832.

Nathaniel Chapman was born in Virginia, a descendant of Sir Walter Raliegh, and attended the Alexandria Academy during boyhood. He started his medical studies at age seventeen at the University of Pennsylvania. Although poor and a stranger, with no acquaintances, he made and kept many influential friends by his gracious and captivating personality.

After two years of medical reading he was graduated in 1801, and after a three year sojourn in Europe he returned to Philadelphia and started medical practice that continued for fifty years. He was an excellent teacher and, in fact, excelled in many things. He was editor of the *Philadelphia Journal of the Medical and Physical Sciences.* It is said that his wit and personal charm were reflected even in his writings.

In 1817 he founded the Medical Institute of Philadelphia, considered to be the first post-graduate school in the United States. In 1847 he became, by popular acclaim, the first president of the American Medical Association. Except for a brief period, he and Dr. Benjamin Rush held the position of Professor of Theory and Practice of Medicine and Clinical Medicine at the University of Pennsylvania for over sixty years.

In 1804 he married Rebecca Biddle, daughter of Colonel Clement Biddle, whom, Mr. Thayer reports, he chose as his future wife even before he met her.

The emergence of the Chapman pitcher after a period of 133 years raises the question as to what happened to the other twelve. Six of the remaining recipients were Fellows of this College. Were their pitchers handed down as family heirlooms or perhaps stored in dusty old attics and forgotten? We do not know the answer, but it is an interesting question.

At the May meetings of the Committee on College Collections and Council, this third memento of Dr. Nathaniel Chapman was accepted with grateful thanks to his great-great grandson, Mr. Sydney Thayer, Jr., and becomes a permanent exhibit in the College of Physicians.

Memoir of Stoughton Ralph Vogel (1907-1966)*

By ANTHONY S. TORNAY, M.D.

STOUGHTON RALPH VOGEL, the only son of Ralph and Pearl Mary (Waller) Vogel, was born on September 7, 1907 at Bryn Mawr, Pennsylvania. He had two sisters, Elizabeth and Catherine. He was named Stoughton after his maternal grandfather and Ralph after his father.

When he was very young the family moved to Germantown where he spent the rest of his life. He attended the C. W. Schaefer Grammar School, a school no longer in existence. At that time his father developed a chronic illness which necessitated a long term hospitalization, and to obtain an education Stoughton had to work in a knitting mill. With effort and perseverance he was able to graduate from Temple University and in 1935 from the Temple University Medical School.

He began his medical career as an intern at Philadelphia General Hospital. After internship he returned to Temple University Hospital as a resident in internal medicine. He received a Master of Science degree in 1940. He began an active teaching career in Temple University Medical School, first as an instructor in medicine, then in 1946 as an assistant professor of medicine, and finally in 1957 as a professor.

When he finished his residency training, he also joined the staff of the Episcopal Hospital as a clinical assistant where he worked in the out-patient department and on the wards of the hospital. In 1943 he entered the U.S. Army Medical Corps, serving as a captain, then major, until his discharge in 1946. Having completed his army service, he returned to Philadelphia and continued his teaching career at Temple University Medical School and his affiliation with the Episcopal Hospital. He became a Chief of Service in medicine at the Episcopal Hospital and was named the Director of the Department of Medicine in 1957, a post he held until his untimely death.

For a number of years his office was at 246 West Walnut Lane. He later moved to 151 West Tulpehocken Street where his tastes were better served. He was interested in gardening and spent much of his leisure time cultivating his flowers. He was also interested in books and his home contained a large collection of books.

From the time he was named Director of the Department of Medicine at the Episcopal Hospital he spent most of his efforts at that hospital. He was interested in people and this interest was demonstrated in the handling of his patients and members of the hospital staff. He was highly regarded by the nurses and other hospital employees. His concern for the welfare of the hospital was so sincere that he seemed to be automatically appointed to every important committee of the staff. Soon after he was made Director of Medicine, he served as the President of the Staff for the two years allowed by the By-Laws of the Staff.

While still a student at Temple University Medical School he became engaged to a young woman who had just graduated from the Philadelphia Normal School. While teaching school, she became ill with an overwhelming pulmonary infection and died. Dr. Vogel never became emotionally involved with any other woman so that he died a bachelor.

He was interested in all aspects of in-

* Prepared and published at the request of the Council of The College of Physicians of Philadelphia.

ternal medicine, as shown by the number of organizations to which he belonged. He was elected a fellow of the College of Physicians of Philadelphia in 1941. He was a Fellow of the American College of Physicians. Some of the other organizations of which he was a member are the American Heart Association, American Trudeau Society, American Geriatric Society, Philadelphia County Medical Society, Pennsylvania State Medical Society and the American Medical Association.

In 1957 he developed a severe episode of dizziness. It was then he found out that he was hypertensive, but he continued to work without respite and did not spare himself. Early in 1965 he accidentally tripped over a hose on the highway and fractured his right radius, the head of which had to be removed. Several months later his back hurt so badly and constantly that he had to undergo surgery for a ruptured lumbar intervertebral disc. He barely recovered from this operation when he suffered a transient episode of aphasia. Despite these three episodes of illness he returned to his duties, but on the morning of January 1, 1966 he suffered a massive intracerebral hemorrhage and died. Philadelphia medicine had lost a valuable son.

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The Albert Einstein Medical Center of Philadelphia: First 100 Years[1]

By J. GERSHON-COHEN, M.D., D.Sc. (MED.)[2]

ONE hundred years, several historical events, the talents of scores of devoted people, and sheer necessity shaped the Albert Einstein Medical Center that stands today. Probably we should date the years from 1864, when the first petition requesting financial contributions to the founding of a Jewish hospital was circulated among residents of the Northern Liberties section of Philadelphia by the B'nai B'rith Lodge. Prior to this time, Philadelphia Jewry had cared for its sick, aged, infirm and destitute in other ways. Its sick were principally cared for in the Pennsylvania Hospital, across the street from the Mikveh Israel Cemetery which had been consecrated in 1740. Burial of the dead and domiciliary care of the aged and ill were entrusted to members of a society specially created for these purposes, among whom Dr. Isaac Hays was most prominently active.

Another mutual aid society, headed by Rabbi Isaac Leeser (Fig. 1) of the Sephardic Congregation of Mikveh Israel, also concerned itself with the welfare of orphans, widows, aged and sick among the Jewish population, which by 1829 was estimated at about 15,000 in the United States, with its main settlement in Cincinnati. Rabbi Leeser hoped for the establishment of a national Jewish hospital which would minister to his co-religionists throughout the land. His wish was realized in part when the first Jewish Hospital in the country was erected in 1849 in Cincinnati to care for the local population of some 5,000 Jews.

In 1852, three years later, the Jews' Hospital (now the Mt. Sinai) was founded in New York to serve a population of some 40,000 Jews. By 1855, when the immigration from revolutionary Germany had reached its peak and many Jews had settled in Philadelphia, the Jewish Foster Home was founded here and sentiment for a Jewish hospital became more resolute. The depression of 1857, however, put a brake on this resolution. Nevertheless, the Hebrew Relief Association came into being and, by engaging two physicians to attend the poor and provide home care and medicines, obviated the need for turning to

[1] Read in a shorter version before the Section on Medical History, The College of Physicians of Philadelphia, 11 October 1966.

[2] Emeritus Director, Department of Radiology, Albert Einstein Medical Center, Philadelphia, Pennsylvania 19141.

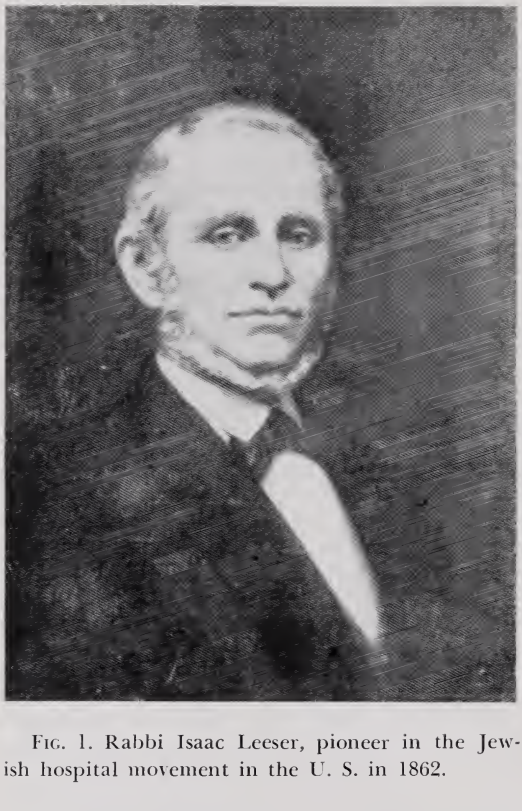

Fig. 1. Rabbi Isaac Leeser, pioneer in the Jewish hospital movement in the U. S. in 1862.

the Blockley Almshouse (now Philadelphia General Hospital), which was not as acceptable as the Pennsylvania Hospital. Kosher diets, of course, could not be guaranteed in either hospital, and the Almshouse was hardly a gentle place to be quartered when one was ill.

With the advent of the Civil War, sick and wounded converged upon Philadelphia, where 15 military hospitals were established. Rabbis and Jewish doctors joined the clergy and physicians of other faiths in offering them succor. Rabbi Leeser, now eager for a Jewish hospital in Philadelphia, joined a recent immigrant, Abraham Sulzberger, the father of one of Philadelphia's eminent jurists, in exploring the idea (Fig. 2). By this time the Philadelphia population of Jews had swelled to 10,000 and a new Reform Congregation, Keneseth Israel, had been established. Unfortunately, ritualistic differences among the Jewish sects hampered united action. Undaunted, in 1863 Leeser and Sulzberger were instrumental in renting a house near 34th Street and Powelton Avenue where sick, destitute civilians could receive medical care. The B'nai B'rith Lodge, meanwhile, strove to create a more harmonious climate in which progress toward a true hospital could be made. This was ultimately achieved and culminated in the petition of 1864 mentioned earlier. Sulzberger was a past president of Lodge No. 3, and this fact—plus a personal charisma and a persuasive approach to the project that overrode his Bavarian accent—no doubt contributed much to the Lodge's sponsorship of the idea and the distribution of the circulars to residents of the Jewish community.

All religious denominations except the Jews had already founded hospitals in Philadelphia. St. Joseph's, located then as now at 16th Street and Girard Avenue, was sponsored by the Catholics in 1849 for the "relief of distressed immigrants from

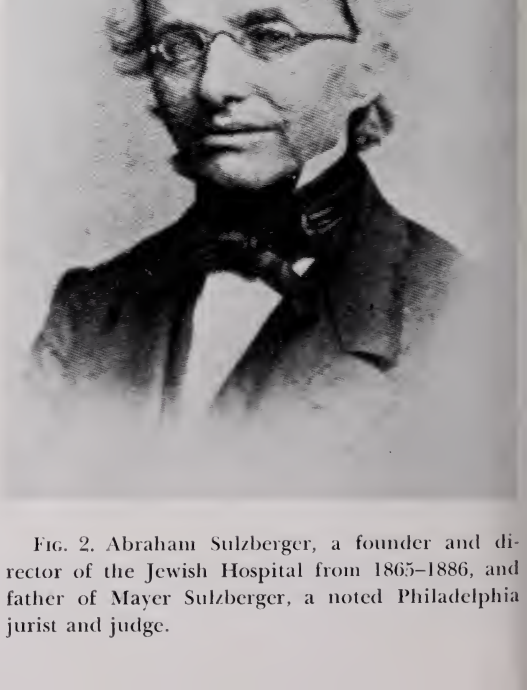

Fig. 2. Abraham Sulzberger, a founder and director of the Jewish Hospital from 1865–1886, and father of Mayer Sulzberger, a noted Philadelphia jurist and judge.

Ireland". In 1852, the Episcopal Hospital opened a "hospital house" at Front and Huntingdon Streets in a section populated largely by immigrants from the British Isles. The German Hospital (now the Lankenau) was chartered in 1860 to care for German-speaking Christians. It was unthinkable that death in any of these institutions should be approached without the administration of last rites by the clergy of these sects. Yet Jews died in hospitals often bereft of the final consolation of fellow-Jew or rabbi because of the poverty and hardship that befell many of these strangers in a new land.

On December 4, 1864, members of the B'nai B'rith took a further step toward establishing a hospital. They organized a committee for the purpose of forming a "Jewish Hospital Association". The first formal meeting of this association was held on Sunday, February 19, 1865 in the National Guards' Hall at 6th and Race Streets. Hundreds of Jewish men and women signed subscription pledges after stirring speeches by Leeser, Sulzberger, and Moses Aaron Dropsie, a prominent lawyer and street railway magnate.

Fig. 4. William B. Hackenburg, president of the Jewish Hospital, 1878–1917.

Fig. 3. Alfred T. Jones, first president of the Jewish Hospital, 1865–1871.

The Association's first president was Boston-born Alfred T. Jones, like Ben Franklin a printer (Fig. 3). He was also president of the Sephardic Congregation of Beth El-Emeth, a Mason, and a member of B'nai B'rith. Isidore Binswanger was named vice-president; Mayer Sulzberger, a young lawyer, succeeded his father as secretary. Prominent among the board of twelve directors were Rabbi Leeser, Abraham S. Wolf, and William B. Hackenburg (Fig. 4).

The building committee at first considered renting a ward in one of the other hospitals as a temporary measure, but this proved fruitless for none was available. When the Civil War ended, a number of government hospitals were advertised for sale and were duly looked into, but none was considered adequate. Finally, suitable

Fig. 5. Woodside Mansion in Hestonville, West Philadelphia, the first home of the Jewish Hospital.

quarters were found in Hestonville, West Philadelphia, consisting of a lovely stone building of 25 rooms located on three acres of beautiful land, known as Woodside Mansion (Fig. 5). In short order the charitable instincts of the Jewish community were mustered to buy the building, contributions coming from rich and poor, from members of the seven congregations, and from the several educational and charitable societies. The hospital site was purchased in September, 1865; the dream was to be realized at last.

Unfortunately, the original subscriptions barely covered the costs of acquisition, and when the first Annual Report was submitted in 1866, the hospital doors had still not been opened to the sick. The wealthier elements of the community were again prevailed upon, with some success, so that, by August 6, 1866, the establishment was ready to admit patients to its 22 beds. Twenty-eight patients were admitted during the first six months of operation, coming under the care of Dr. Adolph Feldstein, a Bohemian physician and a graduate of the University of Prague in 1864. On the recommendation of Dr. Jacob da Silva Solis-Cohen (Fig. 6), Dr. Feldstein was elected for a term of one year to be resident physician and apothecary to the hospital at a salary of $300. Of the some 25 Jewish physicians in the community, Drs. Solis-Cohen, Manly Emanuel, and Benjamin B. Wilson offered their services as consulting physicians without charge. Administration of the little hospital was entrusted to Lazarus W. Kronheimer who, acting as steward (as the post was known in those days), was held responsible for administrative expenditures, kosher diets, behavior of patients, stoking the coal fires, farming, gardening—and milking the cow!

Most of the patients were poor trade peddlers and female domestic servants. The original by-laws stipulated that "no applicant . . . with a contagious disease or one of an immoral nature will be admitted

into the hospital". This injunction was stricken from the by-laws to permit patients with gonorrhea to be cared for.

From the very beginning, the Philadelphia institution, unlike the Jewish hospitals of Cincinnati and New York, was determined to operate on a strictly nonsectarian basis. Alfred T. Jones, in his second Annual Report as president, stated proudly that the hospital "belonged to the community at large". Soon non-Jewish exceeded the number of Jewish admissions, and the hospital with complete impartiality provided free services to all poor and worthy applicants regardless of nationality, color or creed. (The Episcopal Hospital shortly adopted the same practice.) The Jewish Hospital also accepted patients with terminal illnesses, even though they sometimes became wards and their funeral expenses had to be defrayed by the "Hospital Association". Dr. Feldstein was also allotted funds with which to buy suitable clothes for discharged patients whose own attire was not fit for decent society. Thus the first glimmer of a social service function was seen, which later became a formal activity of the hospital.

The annual maintenance costs of the hospital in its early years were in the neighborhood of $25,000. Financial assistance from the small mutual aid societies of the community and from individual contributors was hard to come by during the depression years that followed the Civil War, so in 1869 the "United Hebrew Charities" was organized as a collection and disbursement agency. The Jewish Hospital Association became one of its constituents. The present "Federation of Jewish Agencies" is an outgrowth of this original organization, and today it allots some $600,000 annually to A.E.M.C. toward making up the ever-recurring deficits of operation.

In 1869, the City of Philadelphia announced plans for street extensions which would cut through Dr. Feldstein's living quarters. Remodeling the hospital to circumvent them seemed impractical, so a location for new quarters was sought. A 141½ acre plot of cultivated land was selected on York Road, north of Branchtown, on which stood a mansion, a barn, and a farmer's house. On this site are the present facilities of A.E.M.C. The usual tribulations with fund-raising, however, delayed the dedication of the new "Jewish Hospital and Home for the Aged and Infirm" until September 1873. The new hospital had accommodations for 16 patients in each of two wards, and facilities for a chapel, a reception room, resident physician's office, nurses' residence, apothecary shop, diet kitchens, bathrooms, elevators, and gas lighting (soon to be superseded by electrical facilities). It was regarded as an advanced architectural model of hospital construction by the 22 other hospitals

FIG. 6. Dr. Jacob da Silva Solis-Cohen as a young Civil War officer.

Fig. 7. First building of the Jewish Hospital on Old York Road, its present grounds, in the old Branchtown section of Philadelphia, 1873.

of Pennsylvania (Fig. 7). (Among these, the small Presbyterian Hospital had opened its doors just a few months earlier in July 1872; Hahnemann College had not yet received its papers of incorporation; and the University of Pennsylvania Hospital would not be dedicated for another year.)

The enlarged medical contingent now consisted of Drs. Feldstein, John C. Hall, W. B. Smith, David Davidson, and Joshua R. Evans as attending physicians and Drs. Richard J. Levis and Thomas G. Morton as attending surgeons. A dispensary was added in 1878—by which time the nursing staff had increased from one to two nurses! Anesthesia had been adopted and Listerian antiseptic surgery was being practiced. Specialization was beginning to be encouraged, no longer being condemned as it had been only a decade earlier by Dr. Jacob Solis-Cohen. Surgery and medicine were sharply separated. Surgery at first was limited to amputations and orthopedic corrections, but in time was extended to include operations for hemorrhoids, hernias, and breast cancer, and a little later, to appendectomies. In an era when surgical mortality rates were in the neighborhood of 35%, those at the Jewish Hospital were usually well below this average.

Within a few more years, the aged and infirm were moved to their own new building and special facilities were provided for the chronically ill. In 1900, the "Lucien Moss Home for Incurables" was opened with about 30 beds, most of them taken up with the treatment of tuberculosis.

In 1896, Dr. Edwin A. Jarecki, a Christian, was appointed chief resident physician, a position he held for 38 years (Fig. 8). He introduced the most modern methods of medical procedure and record keeping, and his devotion to duty and loving care of the sick became hallmarks of the professional attainments of a rapidly growing staff of competent men. Among them was Lewis W. Steinbach, who became a noted surgeon under the tutelage of Levis at the Polyclinic Graduate School. Attendants soon included Drs. Francis X. Dercum (neurology), Jay Frank Schamberg (dermatology), DeForest Willard (or-

thopedics), S. MacCuen Smith (otology), and Arthur W. Watson and Louis Jurist (laryngology). The staff was shortly joined by Drs. Henry F. Weber, Solomon Solis-Cohen, Bernard Kohn, David Riesman, William H. Teller, and Moses Behrend, all of whom gave sterling service to the institution for many years. Dr. Behrend's tenure extended from 1899 to 1948, and today he is still actively interested in the affairs of the hospital (Fig. 9).

The X-ray Department was added in 1897, less than two years after Roentgen's discovery. By 1902, a separate surgical building was ready. The nurses' training school, which had started in 1892, expanded so quickly that it acquired its own spacious quarters in the "Eisner Home for Nurses" by 1902. About this time, additional land was acquired, extending the hospital property from 10th Street to York Road. Felix Isman, a prominent city realtor, was very helpful in this acquisition, and it was he who suggested placing at the hospital entrance the six identifying granite columns which had originally graced the front of the U. S. Mint at 13th and Chestnut Streets (Fig. 10).

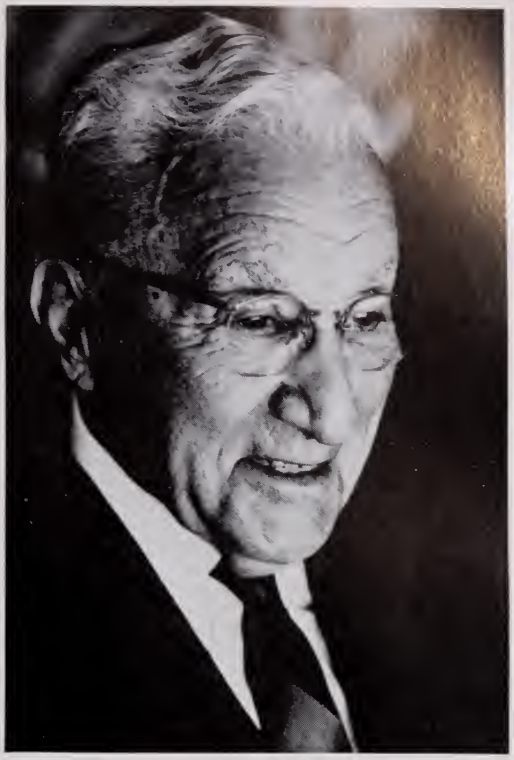

FIG. 9. Dr. Moses Behrend, noted surgeon, affiliated with A.E.M.C. from 1899 to 1948, and in 1966 still actively devoted to the affairs of the institution.

FIG. 8. The tall, handsome Dr. Edwin A. Jarecki (second from the right), chief resident physician and virtually medical director, 1896–1934. He exemplified the spirit of devotion to duty and loving care of patients which was the hallmark of the Jewish Hospital.

Meyer Guggenheim, who started as a poor Philadelphia merchant and became a copper mine tycoon, gave the hospital $80,000 in 1901 to erect a building for private (paying) patients, with the proviso that income so derived be devoted solely to charitable purposes. About this time, too, an isolation ward for contagious diseases and a special pediatrics ward became available.

By 1905, Dr. Jarecki had 5 interns to

Fig. 10. The six identifying granite columns at the hospital's entrance which formerly graced the front of the U. S. Mint at 13th and Chestnut Streets.

oversee and the attendant staff numbered about 190 physicians, including Drs. Benjamin W. Wilson, W. W. Keen, and Orville Horowitz. There were 125 beds for the sick, 35 for incurables, 60 for the aged, and a dispensary to care for some 8,000 patients. The small-sized dream had become a substantial reality.

Funding was, as always, a problem, and the reorganization of the various Hebrew charities into the Federation of Jewish Agencies in 1901 was a great help in underwriting the progressively larger annual deficits. In 1907, with some state aid, a larger nurses' home was built. In 1915, the Kohn family made resources available for a new medical wing. In the summer of 1916, Philadelphia was staggered by an epidemic of poliomyelitis that involved some 27,000 children. Dr. Myer Solis-Cohen, a staff pediatrician, helped to establish the hospital's first research activities under Dr. George D. Heist. Jules E. Mastbaum, following in the tradition of Isman, was so successful in garnering the resources needed that the laboratory was named in his honor. Dr. John A. Kolmer was also prominent in its research activities.

World War I erupted and the hospital's resources were put at the disposal of the government. Training programs for orderlies, nurses, and doctors were vigorously pursued. A large contingent of the professional and nursing staffs entered the military services and served with distinction in practically every theater of the war. In the 1918 Annual Report, the beloved president, William B. Hackenburg, who served the hospital continuously for 54 years, alluded proudly to the accomplishments of the professional staff. His death one month later was a considerable blow to his many friends. He was succeeded by his nephew, Arthur A. Fleisher, who had been an officer of the board since 1902. Mr. Fleisher served as president until the middle of World War II.

In 1920, tubercular patients were transferred to Eagleville Sanatorium, and the Lucien Moss Home restricted patients to those with incurable diseases. The Social Service Department, organized formally in 1916, began to function more actively in providing assistance to poor patients. Under the prodding of Dr. Solomon Solis-Cohen, the medical department expanded rapidly, and cardiology, gastroenterology, and a diabetic clinic soon became active subspecialties. In 1928, a maternity building was built in memory of the Hackenburgs. The administrative affairs were now under the guidance of Alfred Mayer, and Dr. Joseph C. Doane was made the first Medical Director, giving up a similar position at the Philadelphia General Hospital. In 1933, having weathered the depression years following 1929, the hospital was able to erect a six-story surgical building through the

generosity of Mrs. Anna E. Fridenberg. In 1934, the hospital suffered its greatest loss in the death of Dr. Jarecki. His devotion and loyalty to duty since 1896, and the affection he generated among patients and colleagues, probably best exemplify the spirit that characterized the institution throughout its history.

Growth of the hospital continued, the staff constantly expanded, and teaching and research activities became increasingly prominent. World War II interrupted this evolutionary trend as the pattern of service to the armed forces of the first World War was repeated. At this war's conclusion, however, the staff was rehabilitated promptly, and measures were undertaken to create a program with emphasis on teaching and research. The nursing school was again expanded and housed in more adequate quarters, the "David and Mary S. Hirsh Nurses' Home". Judge Charles Klein was now president of the board, and he brought to the attention of the community the inadequacy of some of the buildings.

A survey of the facilities was undertaken by Dr. Henry B. Makover of Columbia University, and his report initiated another milestone in the history of Albert Einstein Medical Center. His recommendations included creation of a system of comprehensive medical care, including preventive medicine, care of the chronically ill, rehabilitation of the disabled, expansion of psychiatric facilties, and care of the ambulant patient—with strong overall stress on education and research.

In 1952, the Northern Liberties Hospital and the Mt. Sinai Hospital were amalgamated with the partial support of the Federation of Jewish Agencies and with the help of several board members, notably Senator Harry Shapiro, Mr. Albert M. Greenfield, and Judge Klein. The Albert Einstein Medical Center, as such, came into existence on February 5, 1952. Mr. Joseph M. First, vice president and general counsel of *The Philadelphia Inquirer*, became the first president of its board.

During the 15 succeeding years, a new "Home for the Jewish Aged" was built on nearby York Road, largely through the dedicated efforts of two prominent Philadelphians, Mrs. Reba Klein and Harry B. Robinson. The Moss Home for Incurables was converted into the Moss Hospital of Rehabilitation; and the (Dr.) Leon Levy Medical and Surgical Building was erected under the able administration of Dr. Pascal F. Lucchesi. Mr. Samuel Daroff was particularly instrumental in garnering the philanthropic funds necessary for these expansions. Physical improvements were made in the Southern Division of the Center, the former Mt. Sinai Hospital.[3] The Northern Liberties

[3] The Mt. Sinai Hospital had its actual beginnings in a four-story furniture factory at 5th and Wilder Streets in 1905, after many trials and tribulations by a few colorful and dedicated physicians practicing in the Jewish ghetto sections of southeast Philadelphia, bordering on the old colonial Society Hill section. The hospital came into being with Jacob D. Lit, a prominent local merchant, as chairman of the board of directors. The motivation to care for the medical needs of a large immigrant Jewish population was as natural as the impulse which resulted in the establishment of the St. Agnes and the Methodist hospitals for adherents of their respective faiths in the neighboring sections of Philadelphia.

When the Mt. Sinai Hospital became the Southern Division of the Albert Einstein Medical Center in 1952, it had grown to an institution with modern facilities for some 350 patients cared for by a large staff of notable physicians.

The Northern Liberties Hospital, located at 808–810 North 7th Street, began operation in March, 1925, after a series of hectic financial struggles which witnessed the demise of the Northern Dispensary after almost 100 years of service and of the Lebanon Hospital after some 25 years of catering to the poor, immigrant, sick residents of that portion of Philadelphia between Vine Street and Girard Avenue and from the Delaware River to Eighth Street. Most of its staff members also served on the staff of Mt. Sinai Hospital. The financial affairs of this institution were always only marginally adequate, and soon after its consolidation as the Eastern Division of the Albert Einstein Medical

Hospital was abandoned. The nursing school was again enlarged and the patient-care program modernized to include services for intensive care, extended care of convalescents, self-care of ambulants and, finally, a rapidly growing home-care program. The out-patient department was enlarged by facilities made available through Samuel Paley, the eldest of the family prominent in television, and the obstetrical-gynecological facilities were housed in a new building made possible by a bequest from Morris Lifter, a successful manufacturer of ice cream.

In 1958, with the resources of his eminent family in the building industry, Mr. Max Wm. Korman helped to create an enlarged research division, which today is expanding rapidly in basic scientific research under the guidance of Dr. Samuel J. Ajl. A cardiopulmonary-renal laboratory was made operative and expanded rapidly. The clinical pathological and X-ray laboratories also set a rapid pace of growth to meet the torrent of advances in scientific medicine. A hospital publication, the *Journal of the Albert Einstein Medical Center,* founded by Dr. Doane, mirrors the bustling activity of the staff, whose work is widely reported in the major scientific journals of the world.

Today the professional complement numbers about 750 members, not including some 20 Ph.D.s and 100 full-time scientific workers in basic research. There are about 85 resident physicians, some 35 interns, and an active teaching program for 3rd- and 4th-year Temple University medical school students.

The little Jewish Hospital that grew into the Albert Einstein Medical Center now appears ready to make another leap forward. Pressure is building up to expand its magnificent facilities into a medical school. The large out-patient department is very busy. There are facilities for 800 acutely ill patients, 200 accommodations for those in need of rehabilitation, 35 in psychiatry, 50 in pediatrics, 100 in obstetrics and gynecology, and the nearby Home for the Jewish Aged serves some 400 residents and has 50 beds for the chronically ill. The associated facilities of the Philadelphia Psychiatric Hospital and the Willow Crest Home for Convalescents are available. Paul J. Johnson, a prominent retired merchant and a recent president of the board, and Earl Perloff, head of a large wholesale grocery organization and the current president, indicated early in 1960 that serious consideration should be given to converting the institution into a medical school—one uniquely structured to meet the challenges posed by this scientific age. The recently celebrated 100th Anniversary, which was high-lighted by two international conferences on cardiovascular diseases and microbiology and attended by physicians from all over the land, pointed up the humane and scientific accomplishments of the institution in the past and indicated some of the avenues open for future growth and development.

Center its facilities were abandoned and its staff integrated with those of the Northern and Southern Divisions.

SUGGESTED READING

1. Whiteman, M.: *Mankind and Medicine:* A History of Philadelphia's Albert Einstein Medical Center. Published by Albert Einstein Medical Center, 268 pp., 1966.

W. Somerset Maugham (1874–1965): A Medical Truant[1]

By FRED B. ROGERS, M.D.[2]

W. SOMERSET MAUGHAM, one of the most distinguished truants from medicine, died on December 16, 1965. Earlier in that year, *The Medical Directory,* whose annual questionnaire he had punctiliously returned since the turn of the century, listed this information about him:

William Somerset Maugham. Companion of Honour. Villa Mauresque, St. Jean-Cap Ferrat, France: Member of the Royal College of Surgeons of England, Licentiate of the Royal College of Physicians of London, 1897; (*St. Thomas's*); Honorary Doctor of Literature, University of Toulouse, 1947; Honorary Doctor of Letters, Oxford University, 1952; Companion of Literature; Honorary Senator, Heidelberg University; Commander of the Legion of Honour.

Storyteller, novelist, dramatist, essayist and world traveler, W. Somerset Maugham was born in Paris on January 25, 1874. His father was legal adviser to the British Embassy there. The family spoke French and the boy learned English, through the roguish humor of the chaplain to the embassy, by reading aloud the police court news in *The Standard.* Maugham's mother died of tuberculosis when he was eight, and his father two years later of heart disease. He was then sent to England and placed under the care of an uncle, the vicar of Whitstable in Kent. The vicar and his wife seem to have misunderstood their frail and sensitive nephew, whose mixture of French and English probably aggravated a stammer which plagued him all his life.

"Willie", as he was known to friends, received his education at the King's School, Canterbury, where he was poor in sports and where the boys and masters laughed at his stammer. Next he spent a year at Hyères in southern France, because of suspected tuberculosis, and at Heidelberg University in Germany, where he studied philosophy. At the age of eighteen, he became a medical student in London, and during his last year at St. Thomas's Hospital he published his first novel, *Liza of Lambeth.* Maugham found preclinical studies dull, but he became absorbed by the work on the wards and in the city slums where his first book was conceived. Upon qualifying as a doctor in 1897, he abandoned medicine and went off to Spain "to become a professional writer".

During the First World War, Somerset Maugham served with an ambulance corps in Belgium and France and then in the Intelligence Department, ending up in Russia which he left at the outbreak of the Bolshevik Revolution. (From the latter experience later came his novel, *Ashenden or the British Agent.*) Owing to hardships he had undergone, he contracted tuberculosis and was forced to spend two years in a sanatorium at Nordrach-on-Dee in Scotland (1918–20). His health restored, he set out on a series of journeys he long had wanted to make, returning to London only when he had received so many impressions that he was "as it were, waterlogged and incapable of absorbing anything more for a while".

Maugham bought a mansion on the

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FIG. 1. W. Somerset Maugham. Portrait study by Karsh of Ottawa.

Riviera in 1927, and the Villa Mauresque, as it is called, became a famed landmark. He spent part of the Second World War in France working for the Ministry of Information, part in London, and part in the United States where he was sent to do whatever he could to help in the common cause. At the close of the war, he returned to the Riviera to his estate "that had been occupied by the Italians and the Germans, shelled by the British Fleet and looted by the French". Carefully restored, the Villa Mauresque became his home again until his death at age ninety-one.

Although he deserted medicine for literature, except for a tour of duty with a Red Cross ambulance unit during the First World War, Maugham apparently never forgot the lessons it had taught him. He often recalled his five years as a medical student. In his revealing book, *The Summing Up* (1938), he wrote: "I do not know a better training for an author than to spend some years in the medical profession. . . . The doctor, especially the hospital doctor, sees life bare".

A prolific author, Somerset Maugham turned out 25 plays, 21 novels and 120 short stories in his long career. He was one of the most popular, successful and versatile writers of this century. "I have put the whole of my life into my books," observed

Maugham in *The Summing Up*. Many of his tales are seen through the eyes of an impartial observer, often a doctor who may be a shadowy self-portrait. As literary models he admired Stendhal, especially his novel, *Le Rouge et le Noir*, and Voltaire, whose style he praised for its lucidity, simplicity and liveliness. In one of his prefaces, Maugham identified two streams of influence on the short story. One was the method of De Maupassant, who constructed pointed tales, with surprising twists of plot (Maugham was dubbed 'the English Maupassant'). The other derived from Chekhov (like Maugham a medical man), who relied chiefly on the evocation of strange characters and atmosphere without significant action. Maugham skillfully varied these techniques to produce his own readable style.

FIG. 2. Maugham at age 18, the year he entered medical school.

FIG. 3. St. Thomas's Hospital and Medical School, London, which Maugham attended.

Maugham retained a lifelong interest in medical affairs. His friend, Dr. W. R. Bett, of London, wrote at his death: "He always liked to hear what was going on in medicine.... Somerset Maugham was the most interesting man I have ever met. I found him urbane and essentially kind, not bored, aloof or waspish. And as a fellow truant from medicine I shall always remember his confession to me: 'Nine-tenths of what I know about human nature I learnt during the five years I spent at St. Thomas's Hospital' ".

"I have seen men since as I saw them

FIG. 4. Villa Mauresque, St. Jean-Cap Ferrat, Alpes-Maritimes.

Fig. 5. W. Somerset Maugham. Oil painting by Graham Sutherland. Tate Gallery, London.

then," wrote Maugham in *The Summing Up*, "and thus I have drawn them.... I have always worked from the living model." Many years later he acknowledged that he had acted foolishly in abandoning medicine so precipitously:

> It was idiotic. Absolutely idiotic. I could just as well have written at night and avoided the desperate financial struggle I had. I am sorry that I abandoned medicine so soon for another reason. The chances of writing anything of permanent value before one is thirty are small. One wastes valuable themes trying to make a living.... The first ten years I wrote, I never earned more than $500 a year. It was a constant struggle against poverty. My books were being published, but I hardly made enough on them to live.

In 1960, the year of his 86th birthday, when he described himself as "an extinct volcano", some two million copies of his books were added to the eighty million then in print. *The Razor's Edge,* his last full-length novel, has sold more than five million copies since its appearance in 1944. *Of Human Bondage,* published in 1915, when its author was forty-one years old, entered literature courses in text editions and has been adapted three times to motion picture film. At least two Maugham characters—Mildred Rogers in *Bondage* and Sadie Thompson in *Rain*—belong to a distinctive fictional company that the world will not forget.

Liza of Lambeth, Maugham's first book, was written and published during his final year in medical school, when he was twenty-three years old. It reflected his service as an obstetrics clerk, during which three week period he attended sixty-three home deliveries. This work led him, pro-

Fig. 6. W. Somerset Maugham. Bronze bust by Sir Jacob Epstein. Tate Gallery, London.

tected by a black bag, "up stinking alleys and into sinister courts where the police hesitated to penetrate".

The plot of *Liza* is a love story. Liza is a young factory worker who lives with her widowed drunken mother in a single room in Vere Street, Lambeth. She has an admirer, Tom, and, though she is not very fond of him, she consents to go with him on the Bank Holiday picnic which the people in the street have arranged. Tom is delighted, but it is his undoing and Liza's, for on the trip they become acquainted with Jim Blakeston and his wife, an older couple who have just moved to the street. Liza promptly falls in love with Jim and a great part of the story concerns their clandestine meetings. They hope, absurdly, that Vere Street does not know about their liaison. It is known, however, and the neighbors subsequently approve Mrs. Blakeston's beating Liza in a street fight. This is Vere Street justice and, as a result of the beating, Liza, after a miscarriage, dies.

Liza of Lambeth created a stir and shocked the conventional society of that day. The senior obstetrician at St. Thomas's Hospital was sufficiently impressed by it to offer Maugham a minor staff appointment; this, he noted, "I unwisely refused". *The Athenaeum* said: "Readers who prefer not to be brought into contact with some of the ugliest words and phrases in the language should be warned that Mr. Maugham's book is not for them". Maugham later wrote: "It was a grand moment for my landlady when she heard Basil Wilberforce, afterwards Archdeacon of Westminster, make it a subject of his sermon one Sunday night in the Abbey. It was a thrilling moment for me when, a week or two later, going to see Mr. Unwin he told me that the first edition was sold out and he was hurrying to print a second". Mr Unwin was a prudent publisher: the young author's cheque was for twenty pounds. This slender success encouraged Maugham and he turned full-time author.

Several years after *Liza,* in 1904, Maugham's novel, *The Merry-Go-Round,* appeared. The chief characters here grapple with the writer's favorite topics: the meaning of life and the nature of reality. Dr. Frank Hurrell, a dominating figure, becomes a spokesman for Maugham's outlook.

A young poet, Herbert Field, is a victim of tuberculosis, a disease which threatened Maugham's life in his teens and after this book. The middle-aged daughter of the Dean of Tercanbury (a disguised Canterbury) falls in love with the poet. When she learns that he is dying and that his only hope of prolonged life is to go to Italy, she is married to him. The Dean is aghast, for Field is socially beneath them. But in the end, when his book is published and the Dean has brought him home to England to die, the cleric is reconciled and develops a deep affection for him. Dr. Hurrell, treating the invalid, says:

> If that man had seen as much pain as I have he'd know that is doesn't refine: it brutalises. It makes people self-absorbed and selfish—you can't imagine the frightful egotism of physical suffering—querulous, impatient, unjust, greedy. I could name a score of petty vices it engenders, but not a single virtue.

In 1907, Maugham's novel *The Explorer,* appeared. This is a swashbuckling narrative of adventure and romance cast in British colonial Africa. An intrepid explorer, Alec MacKenzie, leads an Anglo-African band on a dangerous expedition to stamp out the slave trade in the African interior. The surgeon on this safari is Dr. Adamson, a minor but well-drawn character: "He was an Edinburgh man, with a slow drawl and a pawky humour, a great big fellow . . . ; and his movements were no less deliberate than his conversation".

The surgeon was kept busy tending the malarial sick with quinine and chloral and treating the Swahili wounded in their clashes with Arab slavers from Mombassa.

On their way towards Nairobi, Alec and Dr. Adamson were attacked by blackwater fever. For weeks Alec lay at the point of death.... It was fortunate that he could not be told of Adamson's death.... The good, strong Scotchman had succumbed at last to the African climate; and on this, his third journey, having surmounted all the perils that had surrounded him for so long, almost at the threshold of home, he had sunk and died. He was buried at the foot of a great tree, far down so that the jackals might not find him, and Condamine, with a shaking voice read over him the burial service from an English prayerbook.

Somerset Maugham considered *Of Human Bondage* (1915) his best book. A novel which took two years to complete, its proofs found the author in a dug-out in the Ypres salient during World War I. The title came from a chapter by that name in Spinoza's *Ethics*. In that treatise the seventeenth-century philosopher wrote frankly about the world—facing the facts of power in human society and the various motives of human action.

The story of a young man's gradual awakening to knowledge of himself and the world around him, *Of Human Bondage* is a major literary work of this century—a link between the great tradition of the English social novel and the popular novel of today. In one passage of this book Maugham compares life to a Persian rug, which presents us with exquisite designs and fascinating problems, woven in intricate and alluring colors.

The chief character in *Bondage* is Philip Carey; everything happens to or is seen by him. The story is told in 122 chapters, which are a series of vivid, connected short stories. A long novel, its composition suggests the style of Tolstoy.

Of Human Bondage tells the story of Carey's life from boyhood until he is thirty. It opens with the death of his mother—his father is already dead—and his adoption, as a quiet, shy boy with a club foot, by his uncle, who is vicar of Blackstable (a disguised Whitstable). Philip goes to school with unfeeling sons of gentry at Tercanbury. He rebels against the ecclesiastical career his uncle intends for him and studies painting in Paris until he realizes that he lacks genius. Then, after struggling with poverty and an infatuation for a worthless woman, he settles down as a physician and the husband of a girl, considerably younger than himself, whom he loves but is not in love with.

In *The Summing Up,* Maugham wrote of this work:

It is not an autobiography, but an autobiographical novel; fact and fiction are inextricably mingled; the emotions are my own, but not all the incidents are related as they happened and some are transferred to my hero not from my own life but from that of persons with whom I was intimate. The book did for me what I wanted, and when it was issued to the world... I found myself forever free from those pains and unhappy recollections.

Much of *Bondage* came out of Maugham's medical school years. He explained in the book:

Most of the students took the curriculum of the Conjoint Board of the College of Surgeons and the College of Physicians; but the more ambitious or the more industrious added to this the longer studies which led to a degree from the University of London. When Philip went to St. Luke's (a disguised St. Thomas's), changes had recently been made in the regulations, and the course took five years instead of four as it had done for those who registered before the autumn of 1892.

The first teacher met by the medical students at St. Luke's was the anatomy lecturer, Mr Cameron, "a handsome man with white hair and clean-cut features".

He spoke in a pleasant voice, with well-chosen words, and he seemed to take a discreet pleasure in their careful arrangement. He suggested one or two books which they might buy and advised the purchase of a skeleton. He spoke of anatomy with enthusiasm: it was essential to the study of Surgery; a knowledge of it added to the appreciation of art.... 'You will have to learn many tedious things,' he finished, with an indulgent smile, 'which you

will forget the moment you have passed your final examination, but in anatomy it is better to have learned and lost than not to have learned at all.'

Philip found his clinical years much more interesting than the earlier laboratory ones. As a medical out-patients' clerk he followed Dr. Tyrell, a physician popular with the students.

Dr. Tyrell was a tall, thin man of thirty-five, with a very small head, red hair cut short and prominent blue eyes: his face was bright scarlet. He talked well in a pleasant voice, was fond of a little joke, and treated the world lightly. He was a successful man, with a large consulting practice. ... From commerce with students and poor people he had the patronising air, and from dealing always with the sick he had the healthy man's condescension, which some consultants achieve as the professional manner. He made the patient feel like a boy confronted by a jolly schoolmaster; his illness was an absurd piece of naughtiness which amused rather than irritated.

As a "dresser" in the surgical out-patients' department, Philip followed an assistant-surgeon named Jacobs.

He was a short, fat man, with an exuberant joviality, a bald head, and a loud voice; he had a cockney accent, and was generally described by the students as an 'awful bounder'; but his cleverness, both as a surgeon and as a teacher, caused some of them to overlook this. He had also a considerable facetiousness, which he exercised impartially on the patients and on the students. He took great pleasure in making his dressers look foolish. Since they were ignorant, nervous, and could not answer as if they were equal, this was not very difficult. He enjoyed his afternoons, with the home truths he permitted himself, much more than the students who had to put up with them with a smile.

It was Jacobs who embarrassed Philip by asking him to show his clubfoot to his classmates—an examination which led to the surgeon's correcting the deformity somewhat by operation.

On hospital accident call, Philip felt exhilarated, though fatigued.

The dresser on duty had to be at hand day and night to see any casualty that came in. You were on the move all the time, and not more than an hour or two passed during the night without the clanging of the bell just above your head which made you leap out of bed instinctively. Saturday night was of course the busiest time and the closing of the public-houses the busiest hour. Men would be brought in by the police dead drunk and it would be necessary to administer a stomach-pump, women, rather the worse for liquor themselves, would come in with a wound on the head or a bleeding nose which their husbands had given them: some would vow to have the law on him, and others, ashamed, would declare that it had been an accident.

In his process of maturation, Philip suffered as we might still suffer in our own search for meaning in life. The business of growing up could be as difficult then as now. Philip's solution to his troubles, however, gilded with romance, was a good one. Forsaking the contemptible waitress, Mildred, he came to know and love Sally, a girl whose family he admired, and settled down as a country doctor. Maugham, in all his writings, could not offer a better recipe for the good life.

In a play produced under the title *Caroline* and published as *The Unattainable* (1923), Maugham depicted a physician who perhaps came close to the one he might have been. Dr. Cornish, perceptive and diffident, understands the heroine Caroline better than her two suitors and probably loves her more than either. But when she decides to marry him, Cornish slips gracefully away, not from professional decorum, but because he cannot bring himself to be tied to anyone. This somewhat cynical self-portrait is the one Maugham often left of himself, and his apparently cold recording of such infamous conduct as adultery, bigamy, blackmail and murder irritated his critics but entertained millions of readers.

The Painted Veil, a novel published in 1925, took its title from a Shelley sonnet line: "Lift not the painted veil which those who live call Life". Maugham said that the roots of this tale went back to his student years at St. Thomas's. The model of its hero, Dr. Walter Fane, was Maugham's older brother, Frederic Her-

bert, a lawyer later to become Lord Chancellor of England. The plot combines three of its author's favorite ingredients: travel, fornication, and God.

The novel opens in Hong Kong, and in the first scene Dr. Fane surprises his wife, Kitty, in bed with her lover, Charles Townsend, a charming scoundrel and deputy colonial administrator. The story has melodramatic episodes, but there are incidents likely to confuse soap-opera fans. Fane, whose name denotes a healing temple, is an impersonal bacteriologist—a plodding scientist whose wife married him in a panic against spinsterhood. Hearing of a cholera outbreak at Mei-tan-fu, up the Western River, he volunteers to go there. He tells Kitty:

> The French nuns are doing what they can. They've turned the orphanage into a hospital. But the people are dying like flies. I've offered to go and take charge . . . I am an M.D., you know, and before I specialised I did a great deal of general work in a hospital. The fact that I'm a bacteriologist is all to the good. It will be an admirable chance for research work.

Kitty accompanies her husband to the epidemic where he succumbs to the disease. On his deathbed he forgives her indiscretions. She is befriended by the Mother Superior, who had been born in a chateau and belonged to an ancient French family. When the regenerated Kitty—widowed and pregnant—returns to Hong Kong, she meets the cad Townsend and is promptly re-seduced. Ashamed of this lapse, she returns to her family in England—chastened, hopeful, and feeling nearer to God.

Again in *The Narrow Corner,* a novel published in 1932, a doctor is the central character. The title of this book came from the *Meditations* of Marcus Aurelius: "Short, therefore, is man's life, and narrow is the corner of the earth wherein he dwells."

Dr. Saunders, whose shady activities in his profession have caused his name to be deleted from *The Medical Directory,* settles in China. Here he lives happily in the native quarter of the city of Fu-chou, succeeds in his practice and enjoys an occasional pipe of opium. Skilled as an ophthalmologist, he possesses an ironical outlook on life, a keen sense of humor, and an indifference to other people's conduct. Shrewd and skeptical, he likes good food, drink and comfort, and views with detachment the spectacle of human life.

> Dr. Saunders took an interest in his fellows that was not quite scientific and not quite human. He wanted to receive entertainment from them. He regarded them dispassionately and it gave him just the same amusement to unravel the intricacies of the individual as a mathematician might find in the solution of a problem. . . . Right and wrong were no more to him than good weather and bad weather. He took them as they came. He judged but he did not condemn. . . . He was an agreeable companion, but neither sought intimacy nor gave it. . . . His happiness depended not on persons but on himself.

The adventures of Dr. Saunders in Malaysia, and the people he met there, may have had counterparts in Maugham's travels. The fugitive doctor's philosophy of life is akin to that arrived at by Philip Carey in *Of Human Bondage,* and that expressed by the author in *The Summing Up.* The novelist put so much of himself into Dr. Saunders that *The Narrow Corner* might be considered as somewhat autobiographical. Maugham was in his late fifties when he wrote it and had seen much of life and travel.

The Mixture as Before, a collection of short stories which appeared in 1940, derived its title from a Latin phrase used by the medical profession (*Mistura eadem*). One of these short stories, *Lord Mountdrago,* concerns Dr. Audlin, a psychoanalyst, and a weird experience with dreams and the supernatural. The doctor's patient, Lord Mountdrago, British Secretary for Foreign Affairs, suffers from the neurological phenomena of *déjà entendu* and *déjà fait.*

Maugham was treated by a speech therapist for his stammer, and at one time also sought the help of a psychiatrist for this complaint. It was upon this experience that he based the story of Mountdrago. Dr. Audlin's "intimate conviction that all of their (Freud's and Jung's) theory was hocus pocus", however, was an unusual attitude to ascribe to a psychoanalyst! Of Audlin's analyst role we read:

He had been practising now for fifteen years, and had attained, in the specialty he followed, a distinguished reputation. After all the years during which Dr. Audlin had been treating the diseased souls of men, he knew how thin a line divides those whom we call insane from those whom we call sane. He knew how often in men who to all appearance were healthy and normal . . . , when you tore away the mask they wore to the world, you found not only hideous abnormality, but kinks so strange, mental extravagances so fantastic, that in that respect you could only call them lunatic. If you put them in an asylum not all the asylums in the world would be large enough.

In *Creatures of Circumstance,* a volume of short stories published in 1947, the author's struggle with tuberculosis came forth in *The Sanatorium.* In this tale, Ashenden, the British Intelligence agent, contracted pulmonary tuberculosis and was sent by a London chest specialist to a sanatorium in northern Scotland. Here his skilled power of observation again came into play:

It was a strange world into which Ashenden found himself thrown, when, his health gradually improving, he was able to mix with his fellow patients. There seemed to be a great many people and it took him some time to sort them out. They were of all kinds, young, middle-aged and old. There were some . . . who had been at the sanatorium for years and expected to die there. Others had only been there for a few months.

The physician-in-charge, Dr. Lennox, was small, brisk and genial. He was a good enough doctor, an excellent business man, and an enthusiastic fisherman. When the fishing season began he was inclined to leave the care of his patients to his assistants; the patients grumbled a little, but were glad enough to eat the young salmon he brought back to vary their meals.

After sufficient observation of the disease, Ashenden said:

In certain stages of tuberculosis the slight fever that accompanies it excites rather than depresses, so that the patient feels alert and, upborne by hope, faces the future blithely; but for all that the idea of death haunts the subconscious. It is a sardonic theme that runs through a sprightly operetta. Now and again the gay, melodious arias, the dance measures, deviate strangely into tragic strains that throb menacingly down the nerves; the petty interests of every day and the small concerns are as nothing; pity and terror make the heart on a sudden stand still and the awfulness of death broods as the silence that precedes a tropical storm broods over the tropical jungle.

A romance, leading to marriage, takes place between two patients in *The Sanatorium.* Major Templeton, former soldier-gambler, and Ivy Bishop, a very sick English girl, decide to wed despite the warning of Dr. Lennox concerning the gravity of their common illness. "A cheer was raised," by the patients, "as they drove away, as they drove away to love and death." Ashenden concluded that, "though death is important, it is not so important as love."

Somerset Maugham was a shrewd observer, a cosmopolitan whose literary craftsmanship produced many tales well told. As a medical student he read widely outside of his professional field. During this period, at the age of eighteen, he began compiling a notebook derived from his reading and daily life. From 1892 until their publication as *A Writer's Notebook* (1949), he wrote, "my notebooks amounted to fifteen stoutish volumes". Several anecdotes from his medical school years enter into this collection. Two are quoted below:

At the hospital. Two men were great friends; they lunched together, worked together and played together: they were inseparable. One of them went home for a few days, and in his absence the other got blood poisoning at a post-mortem and forty-eight hours later died. The first came back; he'd made an appointment to meet his friend in

the P.M. room; when he went in he found him lying on a slab naked and dead.

"It gave me quite a turn," he said when he told me.

* * *

The other day I went into the theatre to see a Caesarian.... Before starting Dr. C. made a short discourse.... He told us that the patient couldn't have a child naturally and had had to be twice aborted; but she'd set her heart on having one now that she was pregnant again and though he'd explained the danger to her and said that it was only an even chance that she'd come through, she'd told him that she was prepared to risk it. Her husband wanted it too, and that seemed to weigh with her. The operation appeared to go very well and Dr. C.'s face beamed as he extracted the baby. This morning I was in the ward and asked one of the nurses how she was getting on. She told me that she'd died in the night. I don't know why, it gave me a shock and I had to frown because I was afraid I was going to cry. It was silly. I didn't know her, I'd only seen her on the operating table. I suppose what affected me was the passion of that woman... to have a baby, a passion so intense that she was willing to incur the frightful risk; is seemed hard, dreadfully hard, that she had to die. The nurse told me the baby was doing well. That poor woman.

W. Somerset Maugham left an impressive legacy to posterity. In a literary career extending over more than fifty years he earned a high reputation in four distinct media—the novel, the short story, the stage play, and the sound motion picture film. Nor was that the full extent of his versatility, for he produced books on travel, autobiography, and critical essays on other writers, philosophical and religious beliefs. Commanding the interest of all kinds of readers, he was perhaps more clinical than cynical in his approach. Though he gave up the medical calling early in life, he remained, in his phrase, "an idle apprentice" to medicine. Few truants have been more accomplished in their other pursuits.

Maugham's own declared philosophy was this:

> Our business is right living. The problem of right living is complicated by the fact that there is no one code for everybody; one's job is to find out what is right for oneself and then follow it.

REFERENCES

1. *The Medical Directory 1965: 121st Annual Issue*, London, J. & A. Churchill Ltd., 1965, Part 2, p. 1615.
2. *W. Somerset Maugham (1874–1965)*. TIME, the weekly newsmagazine, Dec. 24, 1965, p. 58.
3. Obituary: *William Somerset Maugham*, British Medical Journal **ii:** 1552–53, Dec. 25, 1965.
4. Obituary: *William Somerset Maugham*, Lancet **ii:** 1350, Dec. 25, 1965.
5. *W. Somerset Maugham (1874–1965)*, Journal of the American Medical Association **194:** 1383, Dec. 27, 1965.
6. Bett, W. R.: *William Somerset Maugham*, British Medical Journal **i:** 365, Feb. 5, 1966.
7. *William Somerset Maugham, January 25, 1874–December 16, 1965*, MD medical newsmagazine **9:** 111–13, Jan. 1966.
8. Lister, John: *Somerset Maugham*. New England Journal of Medicine **274:** 452–453, 1966.
9. *Teller of Tales*, MD medical newsmagazine **1:** 69–71, Dec. 1957.
10. Spiel, Hilde: *The Physician and the Pen: Somerset Maugham*, Ciba Symposium **8:** 133–37, April 1960.
11. Brophy, John: *Somerset Maugham*, London, Longmans, Green & Co., 1952.
12. Jonas, Klaus W. (editor): *The Maugham Enigma: An Anthology*, New York, Citadel Press, 1954.
13. Stott, Raymond T.: *The Writings of William Somerset Maugham: A Bibliography*, London, B. Rota, 1956.
14. Jonas, Klaus W.: *The Gentlemen from Cap Ferrat. With a preface by W. Somerset Maugham*, New Haven, Conn., Center for Maugham Studies, 1956.
15. Pfeiffer, Karl G.: *W. Somerset Maugham; A Candid Portrait*, New York, W. W. Norton, 1959.
16. Cordell, Richard A.: *Somerset Maugham: A Biographical and Critical Study*, Bloomington, Indiana University Press, 1961.
17. Brander, Laurence: *Somerset Maugham: A Guide*, Edinburgh, Oliver & Boyd, 1963.
18. Menard, Wilmon: *The Two Worlds of Somerset Maugham*, Los Angeles, Sherbourne Press, 1965.
19. Maugham, Robin: *Somerset and all the Maughams*, New York, New American Library, 1966.
20. Naik, M. K.: *W. Somerset Maugham*, Norman, University of Oklahoma Press, 1966.

The Treatment by Narcosynthesis of an Acute Traumatic Neurosis[1]

By MELVIN SINGER, M.D.[2]

ON OCTOBER 23, 1965, a thirty year old divorced Negro female completed her usual daily routine as a school crossing guard and returned home to her four daughters, who ranged in age from two to thirteen years. She prepared dinner and settled down to a relaxing evening with her family. She had a boyfriend whom she saw a few times during the week, but her major interests and activities centered around her home and children. During the early part of that evening, the family amused themselves by playing records and dancing. At eight o'clock, she decided to go out to pay an overdue bill at a local store. She promised her childen that she would be gone only a few minutes.

As she walked past her school, a gang of teenagers suddenly encircled, beat, and dragged her to the back of the school yard. There they proceeded to rape, brutalize and ravage her. One of the four hoodlums rounded up the rest of the gang until they numbered thirteen. When they thought they might be detected at the school yard, they dragged the semi-stuporous woman into a car and drove to a nearby deserted house. There, they forced her to submit to sodomy, all forms of other perversions and continuous rape. They also beat her with their fists and burned her thighs, buttocks and genitals with cigarette butts to keep her conscious and obedient. She thought that the only way to save herself was to feign a heart attack and pretend she was dead. This she did, and they ran off leaving her naked on the cold floor.

Dazed, confused, and temporarily blinded, she weakly stumbled out of the house into the city streets. A man driving by saw her, picked her up and took her to a nearby hospital. Within a few hours, she was released and placed in the care of her family. The police were given the names that she remembered hearing during the attack. She and her children were then moved from their home to a distant apartment project to avoid any attempted reprisal from the gang.

Five days later she was admitted to the gynecology service of the Pennsylvania Hospital complaining of a foul-smelling vaginal discharge, lower abdominal pains, dizziness, anorexia and insomnia. Within twenty-four hours, the resident gynecologist realized that she was suffering from more than a pelvic inflammatory disease and requested a psychiatric consultation.

Psychiatric examination revealed an emotionally-responsive, thirty year old, rather large-framed Negro woman of average height. She wore a robe to conceal her bruised extremities and sunglasses to hide her swollen and inflamed eyelids. She tried desperately at first to present a pleasant, cheerful, and cooperative attitude. However, her depressive mood was rather quickly seen behind the veneer, as was her apparent agitation and apprehension. She had a mild nonintentional tremor of the fingers. Her attention and concentration were disturbed and she was easily distracted. She actively tried to keep con-

[1] Kenneth E. Appel Award Essay II, presented by the Philadelphia County Medical Society at a joint meeting with The College of Physicians of Philadelphia, 18 May 1966.

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tion on a superficial level. But after questioning, she admitted that, since the attack, she had suffered from constant tension and anxiety during the day. She tried to keep her mind occupied to help alleviate these discomforting feelings. Nevertheless, she compulsively jumped from one activity to another. The slightest noise would startle her. At night, the most distressing aspects of her symptomatology occurred. Lying in bed, she became increasingly frightened and restless. She imagined that the bed was swinging back and forth in an ever-expanding orbit until she had the illusion it would turn a full circle on its axis. It was as if she were on a ferris wheel in which she would fall to her death when it reached its apogee. The light at the side of her bed was transformed into the black jackets and legs of her attackers. Concomitant with these illusory re-experiences she had auditory hallucinations. She again heard them call each other by name to signal each one's proper turn in their barbaric endeavor. If, finally, she were able to fall asleep, she would quickly awaken, frightened by terrifying nightmares in which the traumatic event was reenacted in all its detail. These experiences had recurred every night since the assault.

She described a lack of interest in her daily chores and activities, anorexia, lightheadedness, and vague lower abdominal cramps. She was worried about her children's welfare. She admitted that her spirits were low, but tried to reverse this feeling. Although she willingly described her symptomatology, her cooperation rapidly vanished when questions were poised about the assault. She tried in vain to eschew any discussion of the attack by changing the subject or repeating her previous remarks. Finally, she related the experience in a sketchy half-joking fashion. Much to her surprise, she already had forgotten most of what had happened. She quickly added how glad she was to be alive to care for her children. Questioned about her feelings toward her assailants, she answered again in a half-joking manner, "I'm just glad I'm alive; I hope they don't hurt my children". Then, with a quizzical look, she asked why she was suffering from these daily and nightly symptoms, failing to connect the traumatic event with its psychopathological sequelae.

In a remarkable manner, her automatic psychic defensive operations attempted to reestablish homeostasis by mastering this noxious insult. Repression, isolation, and reaction formation were clearly utilized to hold the insult in check, albeit unsuccessfully. Thus, at night, more primitive maladaptive measures were needed, i.e., illusion, hallucination and nightmarish re-experiencing of the trauma, to release the pressure which was held down tightly by the lid of repression. Therefore, in her nightly regressed ego state, reality testing was partially lost and archaic projective mechanisms were employed. Her ego's defensive operations, usually sufficient in the past to handle any conflict that arose, this time proved inadequate, and the conflict-free area of her personality was encroached upon and compromised by the struggle. Thus, her memory, thinking, perception, motility, intention, and productivity were all functionally hampered.

One might question whether, without any predisposition, such a severe traumatic event could trigger these symptoms. The literature pertaining to combat neurosis suggests that it could not (1, 2, 4). A normal reaction to a severe trauma during combat consists of nervousness, increased irritability and startle reaction, and insomnia. The presence of any or all forms of neurotic symptomatology, let alone this psychotic-like picture, is considered evidence of an underlying psychopathologic conflict which the individual's defenses were previ-

ously able to handle. Thus, her present conflict drew on the energies bound to her past to inundate the psychic apparatus.

Her past history and premorbid personality supported this view. She was self-sufficient, independent, friendly, hard-working, scrupulously honest and accepting of life's difficulties. She was never able to express anger nor was she known as a very sexually-responsive individual. She divorced her first husband four years after their marriage because of "sexual incompatibility". She was never able to relax during sexual intercourse and many times he was unable to enter her. Only in the last year had she experienced an orgasm, and this occurred with her present boyfriend. He was described as polite, considerate and tolerant of her wishes and demands. Her main interest, however, was in her role as a mother. Even during the assault, she was preoccupied not for her own welfare but for that of her children. Thus, much of her personality and character structure was a reaction formation to pregenital aggression and sexuality. Therefore, she provided fertile soil for a traumatic neurosis to take hold.

Because of the maladaptive effect that her actively operative defenses were achieving, I attempted to loosen the defensive forces with a pentothal interview. I felt that if an abreaction could be accomplished, the bottled-up tensions on her psychic apparatus would be diminished and the defensive operation could reestablish itself, without need for the nightly overflow. She was gradually given 250 mgs. of sodium pentothal as a 10% solution in saline over a two hour period (3). During the first hour, she avoided any discussion of the trauma. Although she finally entered into a discussion of the events leading up to the assault, she was unable to progress into it. She kept blocking at the point at which she left her house. She would repeat again and again the preceding material. Finally, I gently coaxed her to continue. She then slowly, methodically, but intently revealed the experience in all its detail and with an accompanying emotional discharge. In a quite believable fashion and in much less refined language, she wished she could heave her assailants onto a spiked bed, cut their penises off and burn them alive. She cried bitterly as she revealed the incident in its totality. When it was over, she slept. Within twenty-four hours, the resident gynecologist and nursing staff noticed that she apparently was a different person. She was relaxed, cheerful and her tremor was gone. She was able to sleep without interruption. The precedent illusions and hallucinations had disappeared, as had the nightmares. For all practical purposes, the psychopathologic symptoms had vanished. She was discharged three days later. Her symptoms did not return.

During the narcosynthesis, suggestion was not used to remove her symptoms because I feared substitute symptomatology would appear if the psychic apparatus still could not handle the insult after the interview. I also refrained from delving into any feeling of fear, pleasure or guilt that she may have experienced through gratification of her sexual masochistic fantasies. The focus was mainly on relieving the guilt over her anger and allowing the expression of her rage together with the abreaction. I felt that in subsequent interviews, if they were necessary, I might have to deal with those areas left untouched. This proved unnecessary. This remarkable therapeutic result was achieved not only because of the success of the abreaction but also because the treatment was given during the period in which the defenses were actively struggling with the insult and the personality was not yet permanently affected by it. In addition, there was not yet time for the establishment of any secondary gain involving her family, group or so-

and system to contribute to the alteration of her character structure.

REFERENCES

1. Fenichel, Otto: *The Psychoanalytic Theory of Neurosis*. New York, W. W. Norton & Co., Inc., 1945, pp. 117–128.
2. Laughlin, H. P.: *The Neuroses in Clinical Practice*. Philadelphia, W. B. Saunders Co., 1956, pp. 632–686.
3. Linn, Louis: *A Handbook of Hospital Psychiatry: A Practical Guide to Therapy*. New York, International University Press, Inc., 1956.
4. Noyes, A. P.: *Modern Clinical Psychiatry*. 4th Edition, Philadelphia, W. B. Saunders Co., 1954.

Dr. George Glentworth (1735–1792): "A Physician Eminent and Useful"

By RANDOLPH SHIPLEY KLEIN, A.M.*

DR. GEORGE GLENTWORTH, according to his memorialist in the *Columbian Magazine* in 1792, owed his professional success to a regular education in medicine, fondness for reading new medical publications, humanity toward the poor, disinterestedness toward wealthy patients, and unruffled good humor. More than many, he seems to have deserved these encomiums.

Born in Philadelphia on July 22, 1735, George Glenworth served "a regular apprenticeship" to Dr. Peter Sonmans, whose brother-in-law he was to become, and then entered the British army as a surgeon's mate. In that capacity he acquired much practical experience as well as "such a knowledge of men and things as afterwards furnished him with many of those useful and pleasant anecdotes with which he often rendered his conversation instructive and agreeable". This army medical experience alone would have qualified Glentworth to practice physic; but, desirous of further training, he proceeded to Edinburgh. He is said to have received a degree there in 1758, writing a dissertation on pulmonary consumption, but the Edinburgh list of graduates does not include his name. However, he is known to have attended the lectures of William Cullen in 1758–59 (1).

Upon Glentworth's return to Philadelphia, his preceptor, Dr. Sonmans, took him into partnership. In 1761–62 when Dr. Storck, surgeon, oculist, and aurist, who claimed members of the British Royal Family as patients, visited Philadelphia, Glentworth "attached himself to him in such a manner as to be present at all his operations, . . . by which means he became ever afterwards eminent for his knowledge of the diseases of the eyes", and was often consulted by other physicians in such cases (2).

In short, Glentworth was a well-trained, promising young physician when, in 1766, he joined Dr. John Morgan and others in founding the Philadelphia Medical Society. On November 4, 1768, the entire membership of the Medical Society was voted into the American Society for Promoting and Propagating Useful Knowledge, held at Philadelphia, which, a few weeks later, united with the American Philosophical Society, to form the American Philosophical Society, held at Philadelphia, for Promoting Useful Knowledge, which still exists. To the American Society Glentworth had already presented, through Dr. Morgan, samples of Labrador tea, which several botanists present pronounced to be a species of *Cassina*. In the enlarged American Philosophical Society he indicated his desire to be considered a member of the Committee on Medicine and Anatomy and on Husbandry and American Improvements; but, he attended only two meetings, in the spring of 1769, and never appeared again (3).

During the next few years, before the outbreak of the War for Independence, Dr. Glentworth continued and extended his practice. In January 1773, pointing out that "the dreadful Havock that the Smallpox daily maketh . . . is so severely felt . . . that every Attempt to elude the Rage of this malignant Disease merits Encouragement from all the Friends of Mankind", Dr. Glentworth provided "a convenient House in this City, with proper Accommo-

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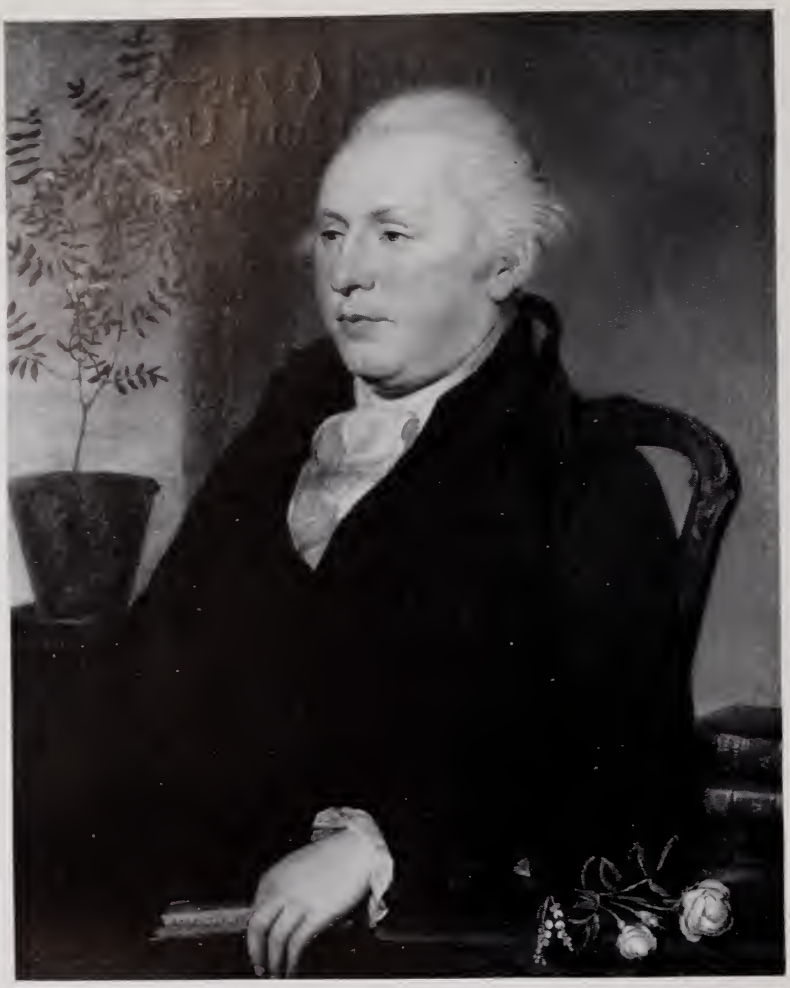

Fig. 1. Portrait of Dr. George Glentworth by Charles Willson Peale, reproduced through the courtesy of Independence National Historical Park.

"The recently-discovered signature and date, 1794, shows that this can only be a posthumous portrait of Dr. Glentworth, and suggests that his son, the equally eminent physician, Dr. Plunket Fleeson Glentworth, may have posed for the likeness." Quoted from Charles Coleman Sellers, *Portraits and Miniatures by Charles Willson Peale,* Transactions of the American Philosophical Society, Vol. 42, Part 1, 1952. (*Editor's Note.*)

dations and a tender and skilful Matron to superintend it". In his public advertisements the doctor tried to overcome popular prejudice against inoculation by publicizing the endorsements of "the most respectable Personages in Europe" and referring to the encouragement the method had received from "Gentlemen of Eminence in the Faculty of Physic, in different Cities of America, where Inoculation is tolerated" (4).

Socially as well as professionally, Glentworth was accepted in Philadelphia. He was a member of the Fishing Company of Port St. David's in 1763, as was Sonmans; and, in 1772, he was one of the original members of the Society of Sons of St. George, whose members flaunted their English background as others did their Scottish, Irish, Welsh, and German origins (5).

During the Revolutionary War Glentworth served several years as a military sur-

geon. Informing his patients that he was leaving them in the care of "a gentleman of skill and assiduity, who dwells at his house", Glentworth left Philadelphia in July 1776 to attend the sick and wounded of the Flying Camp in the Jerseys (6). He took his apprentices with him, and used his entire supply of medicines in the army's service. On April 10, 1778, he was appointed a regimental surgeon, and the next year was senior surgeon of the General Hospital. He took the oath of allegiance on February 23, 1778. During the suspension and court-martial of Dr. William Shippen, Jr., Glentworth was director-general of the Hospital in the Middle Department. In 1780 he caught hospital fever; for a time his life was despaired of; and, upon his recovery later in the year, he returned to private life (7).

His compensation while in the army had never been all Glentworth believed he was entitled to. Accordingly he and two other supernumerary physicians and surgeons of the General Hospital appealed to Congress on June 22, 1781, for depreciation half pay, on the same principle which was applied to supernumerary judge advocates, regimental surgeons, and chaplains. The Secretary of War recommended that Pennsylvania settle the account on the principles suggested in the memorial. This request not being fulfilled, Dr. Glentworth petitioned the United States Congress in 1791, describing his services from 1776 to 1780, and explaining that he had not received sufficient compensation (8).

Back in private life, Glentworth resumed his practice. In 1779 he had been elected to the staff of the Philadelphia Almshouse and continued in the post two years; in 1782 he was appointed one of the inspectors of the Humane Society, whose purpose was to rescue persons from drowning and other premature accidental deaths; and, in January 1787, he became an original member of The College of Physicians of Philadelphia. Though he had never displayed much interest in the American Philosophical Society, beyond attending a meeting or two and giving £5 worth of lumber toward the building of the Society's Hall after 1785, he had a keen interest in natural history. His garden was notable for "a numerous collection of beautiful bulbs and other flowers", which he kept up by annual importations from Holland, and Dr. Johann David Schoepf, the German physician and botanist, pronounced Glentworth one of the few "florists" in America. Appropriately Charles Willson Peale painted his portrait with a pot of flowers in one hand and a bunch of roses on the table near the other. Glentworth also presented to the historical and natural history museum of Pierre E. DuSimitière a stalactite from a cave near Millerstown on the Susquehanna in Pennsylvania and a stone spearhead (9).

Dr. Glentworth married Margaret Linton of Philadelphia in 1764, and the couple had two sons, Peter Sonmans Glentworth and Plunket Fleeson Glentworth. Both followed their father into medicine. The first died in the Philadelphia yellow fever epidemic of 1793; the second, elected to The College of Physicians of Philadelphia in 1792, had a long career as a practicing physician.

Dr. George Glentworth died peacefully on November 4, 1792, of "a misplaced gout", and was buried two days later in St. Paul's burial ground, Philadelphia. Dunlap's *American Daily Advertiser* paid tribute to him: "As a physician he was eminent and useful; and, to a fine genius, joined an excellent heart" (10).

REFERENCES

1. An Account of the late Dr. George Glentworth. Columbian Magazine, Vol. II, 367–368, 1792.
2. *Ibid.*
3. Gilbert Collection, Manuscript Letters, III, 289, The College of Physicians of Philadelphia; American Society, Minutes, 11 March 1768; George W. Norris: *Early History of Medicine in Philadelphia,* pp. 115–116, Philadelphia, 1886.

4. Pennsylvania Packet, 4 January 1773.
5. *History of the Schuylkill Fishing Company,* pp. 401–402, Philadelphia, 1889.
6. Pennsylvania Evening Post, 18 July 1776.
7. Pennsylvania Archives, Series 5, Vol. II, 767; Miscellaneous Treasurer's Accounts of G.A.O., 1790–1894, Independence National Historical Park; Journals of the Continental Congress, 2 May 1777.
8. George Glentworth's Memorial to Congress of United States, 17 January 1791, Society Collections, Historical Society of Pennsylvania; Journals of the Continental Congress, 22 June 1781, 3 January 1782.
9. Pennsylvania Magazine of History and Biography, Vol. LVII, 44, 1933; Pennsylvania Packet, 7 September 1782; George W. Norris: *Early History of Medicine in Philadelphia,* p. 118, Philadelphia, 1886; Johann David Schöepf: *Travels in the Confederation,* Vol. I, p. 95, Philadelphia, 1911.
10. Dunlap's American Daily Advertiser, 12 November 1792; George Glentworth Will, 6 November 1789, Register of Wills, Philadelphia; Lyman H. Butterfield (Ed.): *Letters of Benjamin Rush,* Vol. II, p. 658 n, Princeton, 1951; Pennsylvania Archives, Series 2, Vol. II, 115.

Memoir of Edward Bell Krumbhaar 1882–1966*

By ESMOND R. LONG, M.D.

EDWARD BELL KRUMBHAAR, a life-long benefactor of The College of Physicians of Philadelphia, a member of the College for 42 years, and its President from 1940 to 1943, was born in Philadelphia on August 1, 1882, and had the primary years of his education in that city. He later attended the Groton School in Massachusetts and was graduated from Harvard University in 1904. With an unusually good educational background thus acquired, he entered the School of Medicine of the University of Pennsylvania with a mature appreciation of scholarship and research. When he was graduated in 1908, he had already contributed significantly to the field of medical history and investigation.

Well trained in pathology by the time he was graduated in medicine, he became the resident pathologist at the Pennsylvania Hospital in 1908, an institution rich in tradition and eminent among the oldest and most distinguished hospitals in the United States. There Krumbhaar came under the guiding influence of Warfield T. Longcope, Director of the Ayer Clinical Laboratory of the Hospital, who was later to become one of this country's most noted internists. Throughout his life, Krumbhaar acknowledged a formative and guiding influence from Longcope. Longcope started him on research problems in the physiology and pathology of the heart, for which Krumbhaar became famous, and, even more importantly, endowed him with a spirit of inquiry and modesty in medical research that characterized him for the rest of his life.

Longcope stimulated Krumbhaar to commence studies of the Bundle of His, and Krumbhaar applied his knowledge in that field to a timely investigation of a case of Stokes-Adams disease brought to his attention by Alfred Stengel, one of his teachers at the University of Pennsylvania. In connection with these studies, Krumbhaar worked up the history of heart block in its physiological and pathological aspects in extraordinary detail. From this piece of historical research we may date Krumbhaar's overriding interest in the history of medicine, which was exhibited in every project in medical research on which he worked from then on.

For more than twenty years Krumbhaar was intensively engaged in studies, first, of the functional and pathological disorders of the heart and, then, on diseases of the blood-forming system. He was a pioneer in use of the string galvanometer at a time when, as he himself said, "solvable problems were tumbling at our feet". He published his first paper in that field in 1916. It is of interest to note that some 35 years after these early electrocardiographic studies, he collaborated with associates in an investigation of the ballistocardiograph. In the other domain, that of the hemopoietic system, he became particularly noted as an authority on diseases of the spleen. His technical contributions in this field were outstanding. Characteristically he began them with a historical study on extirpation of the spleen.

His paper on that subject was read in 1914 before the Section on Medical History of The College of Physicians, a section

* Read before The College of Physicians of Philadelphia, 5 October 1966. Prepared and published at the request of the Council of The College of Physicians of Philadelphia.

which Krumbhaar as a medical student had actually helped to organize and with which he was long intimately concerned. And at this point I should say that this memoir will be concerned chiefly with Krumbhaar's long and mutually productive relations with The College of Physicians. His life was a diversified one, with notable contributions in numerous fields. In the space ordinarily allotted to biographical memoirs of our members, it is impossible to do justice to his contributions in all his fields. Fortunately, other biographical records are being published, with emphasis on contributions in other relations. So it is not out of order here to list those contributions briefly and dwell with more leisure on Krumbhaar and The College of Physicians.

We may note in this brief review that Krumbhaar passed through the earlier grades in the Department of Medicine of the University of Pennsylvania, served with distinction under Richard W. Pearce in the Department of Research Medicine of the University, where some of his best work on the spleen and anemia was carried out, and became Professor of Pathology and head of the department of that name in 1927—a position he held for twenty years, before his retirement as emeritus professor. In 1928 he was President of the American Association of Pathologists and Bacteriologists, and in subsequent years president of most of the other pathological societies of this country, including the old and tradition-rich Philadelphia Pathological Society. In that position he presented the first William Wood Gerhard Medal of the Society to William H. Welch, who spoke eloquently on Gerhard and his founding role in the Philadelphia Society three-quarters of a century before. In 1957, with characteristic simplicity, Krumbhaar gave one of his last talks before the society, entitled modestly, "The Worthy Past of Our Society".

Briefly we may note Krumbhaar's directorship of the laboratories of the Philadelphia General Hospital (1920–27), his service as a medical officer in the American Expeditionary Force in France in World War I (1917–19), his service as consulting pathologist to several hospitals, his membership in many American medical societies of distinction, and his honorary fellowship in the Royal Society of Medicine in London.

One of the most notable services of all was Krumbhaar's editorship of the *American Journal of the Medical Sciences*, which occurred in a distinguished line of editors, all of them incidentally members of this College. He succeeded Nathaniel Chapman, Isaac Hays, Isaac Minis Hays, E. D. Davis, Alfred Stengel, Francis Packard, A. O. J. Kelly, George Piersol, and John H. Musser, and more than maintained the scholarly supervision of this periodical, called by Sir William Osler "one of the few great journals of the world".

Krumbhaar's early participation in talks leading to the formation of the Section on Medical History of The College of Physicians has been noted.[1] He read many papers before this section, which remain as valuable sources of reference. A noteworthy one was "The Early Days of the Oldest Medical Monthly, the *American Journal of the Medical Sciences*" (1929). Krumbhaar was a great admirer, and a personal friend, of Sir William Osler. He wrote a paper on Osler's connection with The College of Physicians of Philadelphia, which emphasized Osler's interest in the pathological collections of the College.

Few people, as much as Krumbhaar, have stressed the value of the College's great collections for research on the achievements of the past. Few, it may be

[1] Krumbhaar's literary contributions, and service to The College of Physicians of Philadelphia, have been described in a highly informative paper by Samuel X. Radbill: Transactions and Studies of The College of Physicians of Philadelphia, (4th Series) Vol. 25, No. 1, 35–40, June 1957.

said also, have done as much to put the present in historical perspective with the past. He himself contributed notably to those collections, with gifts of World War I material, archival material from the Department of Pathology of the University of Pennsylvania, correspondence in connection with the *American Journal of the Medical Sciences*, and records of the Council of the College itself, which might otherwise have been lost. He aided the construction of many exhibits in the College and made a special point of using them to stimulate medical students in the history of medicine. Fittingly there is today a student award, the Krumbhaar Award, presented annually by the College's Section on Medical History to a medical student for accomplishment.

Krumbhaar had many warm friends. Notable among these was Francis Packard, distinguished Philadelphia otolaryngologist, president of the College from 1931 to 1934, editor of the *American Journal of the Medical Sciences*, and in particular, with reference to Krumbhaar's early interest in the history of medicine, editor of the *Annals of Medical History*. Krumbhaar became an associate editor and contributed many articles to that *deluxe* and scholarly journal, and, when it became necessary for the journal to suspend publication, he wrote a warmly appreciative note on the service of Dr. Packard in advancing the study of medical history.

These two truly remain immortal in the College. Each was a constant and discriminative collector of rare books and fine first editions. Each was generous, with continuous solicitude for the Library of the College of Physicians. One of the most valuable elements of the Library today is the Packard-Krumbhaar alcove, a rich collection of the volumes they contributed to the College.

In the College and out of it, Krumbhaar, whose talent as pathologist, editor, and medical historian, was equalled by his skill as an administrator, published voluminously in the field of medical history. In a sense the College served as a base of operations. He attended many international congresses on medical history, and brought them to the attention of Americans interested in the field. With the example of the international congresses before him, he set out to organize an American association with similar aims. In 1924 he brought together in The College of Physicians of Philadelphia a group of men who might be interested in such an association. The actual organization of the group took place a year later, with Krumbhaar in the responsible position of secretary, and the eminent historian, Fielding Garrison, as president. Krumbhaar was later elected president, and in 1939, in recognition of his founding role, was made honorary president for life.

An account of Krumbhaar's literary contributions to be adequate should note much more, including 1. his translation, revision and extension of Arturo Castiglioni's "History of Medicine", 2. his editorship and authorship in a series of highly useful short compendia of the history of numerous branches of medicine, brought together under the title "Clio Medica", 3. his many skillful and perceptive book reviews, and 4., as important as any, his obituaries and biographical memoirs. He had a wide acquaintance among great men in the sciences in which he was himself competent, and his tributes to them in the pages of medical and historical journals have preserved the essence of their lives in a way that few could match.

Krumbhaar died after some months of illness at his home in Chestnut Hill, March 18, 1966. While he attained abundant recognition for his accomplishments in the fifty years during which he was most active, he was himself modest, as well as warmly friendly, and never sought fame. His aim was scholarly service to the causes he espoused.

He is survived by his wife, Helen Dixon

Krumbhaar, and their two sons, Peter and David. It should be noted here that in the field of his early choice, blood dyscrasias, Krumbhaar and Mrs. Krumbhaar, who was also in military service in World War I, carried out an important study that led to a paper on "Blood and Bone Marrow in Mustard Gas Poisoning", which was documented in a well illustrated article with a wealth of histological detail.

In the quiet surroundings of their home in Chestnut Hill nearly fifty years later, Krumbhaar died in his 84th year. He worked up to the time of his death. His last book, "Isaac Cruikshank, A Catalogue Raisonné, with a Sketch of His Life and Work", was finished shortly before he died, but came off the press after his death—a not uncommon event in the lives of men who never stop being productive.

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Presidential Address: The College of Physicians of Philadelphia*

By JOHN H. GIBBON, JR., M.D., SC.D. (HON.)

Fellows and guests of The College of Physicians of Philadelphia, it is with a sense of sadness, mingled with relief, that I rise to give my last annual address as President. The sadness, and the relief, are because I end my tenure of the office of President tonight. The past three years as your President have been among the happiest and fullest of my life. Working with the dedicated Fellows and employees of this grand old institution has been a source of intense and serene joy to me. From now on my responsibilities will be greatly diminished, but my devotion will remain.

The skeleton of this institution lies in its bricks and mortar and its marble halls housing an immense collection of periodicals, books, manuscripts, portraits, works of art and a medical museum. However, the life and blood of the institution now, as always, lies in people and their ideas, and of these I shall speak.

May I first of all extend my heartfelt thanks to all the Fellows and employees of the College who have worked so hard over the past three years to nurture the growth of good things for the future and yet to preserve the beauties and traditions of the past.

* Annual Address of the President, The College of Physicians of Philadelphia, 4 January 1967.

Long overdue, a Planning Committee was appointed last Spring, and, under the able Chairmanship of *Donald Pillsbury,* has engaged in an intensive study of all the activities of the College and its library. Their work is continuing and I am sure that the recommendations of the Committee will do a great deal to move the College forward in the right direction.

The second individual I should like to refer to is *Fred B. Rogers,* who terminated ten years of service as Editor of the Transactions on April 30, 1966, because of the press of other duties. He has been succeeded by *Robert E. Jones,* who assumed the duties of Editor on July 1, 1966. Our Secretary, *Bill Steiger,* who has so long and faithfully served the College, resigned this past summer because of his additional duties as Chairman of the Department of Medicine at Temple University. *George Hahn* agreed to serve as Secretary *pro tem.*

George Allen, the former superintendent of the College, died recently and the Secretary of the College wrote his family a letter of condolence. Our devoted Executive Secretary, *Miss Lang,* slipped on the marble floor of the Hall of the College at the November 2, 1966 meeting and fractured her forearm. Many of her duties have been assumed by *Miss Fritz,* working

overtime aided by *Miss Huber,* and in financial matters by *Mr. Eldredge. Mr. Elliott Morse* continues to carry on his Herculean task of acting as Librarian of the College, soliciting funds for the College Library, drawing up plans for grant applications, and serving on both the Library and the Hall Committees. How he manages to accomplish all that he does, I am really unable to explain. *Mr. Kolodziejski* has continued to do a most outstanding job as Superintendent. He has replaced the ceiling lights in the Mütter Museum at a cost considerably less than any of the submitted bids for the work.

Kendall Elsom has continued his efficient and devoted service this past year as Treasurer and a member of the Finance Committee. *George Blumstein* has revitalized the functioning of the Finance Committee as its Chairman and has also continued to work miracles as Chairman of that one-man Committee, the S. Weir Mitchell Associates, which as the only significant fund-raising activity of the College in recent years has added almost $150,000.00 to the endowment of our Library.

Last, but by no means least, I should like to thank *Fran Wood* for all his help to me over the past three years. He has ably presided over meetings of the Council and of the College in my absence. He has been a valued friend and Counselor and his mind has been fecund with plans for the good of the College. In handing over to Dr. Wood the reins of office of the Presidency, I do so with the firm conviction that he will breathe new life into this venerable institution and guide it with a firm hand through whatever tempests may lie ahead.

Somehow ideas have been burgeoning around the College for the past year. Perhaps it is because the Officers of the College and the Council indicated that they were interested in ideas and would be receptive to them. At all events, we have had floods of suggestions as to how to make better use of the College and its facilities and how to make the College better known in the City of Philadelphia. Someone has even suggested the formation of a private club for Fellows with a bar, lounge and restaurant in the College building. Our former Secretary, Dr. Steiger, has made suggestions for streamlining the meetings of Council, eliminating routine, unimportant business so that the Council can devote its attention to more important matters.

Of all the ideas which are giving new life to this old institution, I would say that, in the minds of the Planning Committee and the Council, the most important one is to obtain a paid Executive Director for the College. All of us concerned have been searching for a person to fill this job of Executive Director. The position has been offered to several people who, for one reason or another, were unable to accept. The search is continuing. I believe that the second most important idea is that the College should have a permanent Women's Committee. Apparently no organization can be a success nowadays without the backing of women behind the scenes, or out in front, which is where we hope to have them!

Finally, I would like to express my firm conviction that The College of Physicians of Philadelphia, which was founded 180 years ago on January 2, 1787, can look forward to continuing to play a dynamic role in the medical community of the Delaware Valley and in the Nation for many years to come.

Organelle Pathology of the Liver[1]

By HANS POPPER, M.D., PH.D.[2]

HISTORICALLY, the *pathology of organs* goes back to the fathers of medicine, Hippocrates and Galen. It was brought to bloom when Bichat localized disease into individual tissue and when Antonio Benivieni's *De abditis nonnullis ac mirandis morborum et sanationum causis* appeared in 1507. It reached its peak in 1761 with Giovanni Morgagni's *De sedibus et causis morborum per anatomem indagatis.* In the middle of the last century Virchow developed *cellular pathology* which has contributed so much to the knowledge of disease. Alterations of the cell became the basis of the tremendous advances in our understanding of the biology in medicine in the past 100 years. Morphologic fine structural techniques, particularly electron microscopy and cell fractionation, singly and together, detected processes on ultramicroscopic interphases. This led to the molecular biology, probably the most productive line in recent biologic research. From this developed molecular pathology, apparently initiated by Linus Pauling's concept of sickle cell disease. Molecular pathology uses the highest resolution of electron microscopy on a level where the distinction between function and structure disappears. There remains, however, a gap between molecular and cellular pathology in which finds itself the malfunction of cellular components, i.e., the organelles. These represent specialized units capable of carrying out defined physiologic functions. They are cell compartments, with their phospholipid membranes recognized by low resolution electron microscopy. The morphologic alterations of organelles of viable cells in man may explain human disease when cellular pathology has failed us. Moreover, organelle pathology offers a potential tool for diagnosis.

In the following, I shall attempt to survey the still embryonal organelle pathology of the liver. Study of the anatomy of the liver probably goes back further than that of any other organ, because its lobulation in sacrificial animals was used by the Babylonians to foretell the future and in ancient Rome the haruspices predicted the outcome of a war by examining the liver. Glisson initiated the anatomical pathology of the liver and Malpighi of Bologna opened up its normal and abnormal histology. Rokitansky described as acute atrophy its complete destruction in what we now call viral hepatitis.

Diffuse degeneration of the organ without necrosis as seen on the autopsy table in intoxications and infections was designated as "cloudy swelling" without any correlation between functional impairment and structural alteration. As early as 1928 the German pharmacologist Hoppe-Seyler recognized increased protein content in cloudy swelling (1). Subsequent decades considered the diffuse involvement a toxic hepatitis while, since World War II, the hepatitis virus has been held responsible for many diffuse hepatic injuries, icteric or anicteric, in which no other cause was demonstrated. Today environmental factors such as alcohol abuse, drugs and even fungal and plant toxins are coming into the foreground.

In the liver, an organ sensitive to oxygen deprivation and particularly rich in proteolytic enzymes, many changes occur in the agonal period or after death. Liver

[1] Alvarenga Prize Lecture XXVI, The College of Physicians of Philadelphia, 7 December 1966.

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biopsy subsequently permitted the study of the liver during life without the interference of the processes just mentioned. Here the emphasis was placed upon cellular alterations, more frequently those of inflammatory cells than of hepatocytes. The correlation between the histologic picture and functional alterations, however, remained poor because histologically impressive lesions include necrosis of liver cells, or excess deposition of material readily demonstrable like fat or of abnormal tissue components like alcoholic hyaline, or, finally, inflammation. Physiologic investigations have indicated that removal of a greater part of the liver is not associated with a significant deficit in hepatic function and seldom is the loss of liver cells by necrosis extensive enough to account for a hepatic functional deficit. Similarly, abnormal deposition involves only few cells and it is questionable whether steatosis produces functional abnormalities (2). Statistical analysis of liver biopsy specimens rather revealed that diffuse alterations of the apparently viable liver cells correlate best with impaired liver cell function (3). These are represented by subtle features that greatly depend on histologic processing, such as variation in extent and staining qualities of the cytoplasm. These spurious alterations are used only with difficulty in biopsy diagnosis, but are reflected clearly on fine structural analysis, particularly electron microscopy, which illustrates changes of the organelles well correlated with functional alterations. However, the attempt to correlate one such alteration in man and experimental animals with one etiologic agent has so far failed (4).

It deserves emphasis that, similar to the response of damaged cells, the organelle response to injury also consists of a series of processes which include catabolism, anabolism, repair or cleansing, and regeneration. The first morphologically recognized hepatocytic manifestations are catabolic changes of the endoplasmic reticulum (ER) and of the ribosomes in most experimental and clinical circumstances, with human viral hepatitis representing a well studied example (5). In this group of organelles the key functions of the liver are localized. The ER consists normally of parallel profiles around slit-like spaces. They are either lined by ribosomes, arranged usually in a helix-like fashion to form polysomes, and they are then designated as rough, or they are devoid of these ribosomes and are called smooth. The polysomes are the site of protein formation and they account for the basophilia of the hepatocytes (6). They may be free in the interorganelle fluid called hyaloplasm and then are related to the synthesis of structural protein. By contrast, the synthesis of the proteins secreted by the hepatocytes is localized to the ER-associated polysomes. These proteins include, besides the various coagulation proteins, albumin, alpha- and beta-globulins and the low density alpha-lipoproteins, the last being the vehicle for the hepatocytic secretion of triglycerides. In early injury of the viable cells, the rough ER is shattered and vacuolated and this is associated with depression of protein secretion (7). This is reflected functionally in a delayed appearance of the label in serum proteins after tagged amino acids have been given. At the same time, lipid droplets may appear in the ER (liposomes) (8, 9) as an initial step of the fatty metamorphosis accompanying many liver injuries and related to faulty secretion of triglycerides as low density alpha-lipoproteins. It is today not decided whether this represents impaired formation of lipoproteins or inadequate combination of apolipoprotein with triglycerides (10) or, even more probably, faulty release of the lipoproteins from the ER. While the alterations of the rough ER are correlated with suppressed hepatocytic protein secretion and probably with clinical manifestations such as malaise or bleeding tendencies, this does

not hold true for all other functions. The ER is the site of glucose-6-phosphatase, the enzyme responsible for blood glucose formation. Although the glucose-6-phosphatase reaction decreases on histochemical analysis in mild liver cell injury, hypoglycemia occurs only in the most severe forms.

Eventually, in any type of cellular damage, injury of mitochondria occurs, morphologically reflected in enlargement, alteration of their cristae and deposition of crystals. Functionally, the disturbance is manifested by alteration of oxidative phosphorylation, which means interference with the energy supply to the cell. Mitochondria are the primary target in oxygen deficiency, when the mitochondria enlarge, and peculiarly enough also under increased oxygen pressure, for instance, in hyperbaric chambers or in space capsules (11). Mitochondrial changes are also one of the early effects of sustained alcohol administration, when human volunteers receive for several days barely intoxicating doses of alcohol. Under these circumstances, the enlargement and deformity of mitochondria even precede steatosis (12). In alcoholic liver disease (13) deformity of the mitochondria is conspicuous. This raises the possibility, also claimed as result of experimental studies (14), that the hallmark of human alcoholic liver injury, the alcoholic hyaline of Mallory, represents altered mitochondria. However, it is not possible to trace the origin of these peculiar bodies on the basis of their morphologic appearance. The functional significance of the mitochondrial injury in alcoholics is not yet established. It possibly accounts for the sudden death of alcoholics.

Damage of the cell or plasma membrane is another alteration of what may be called a specific cellular organelle. An extensive portion of the surface of the hepatocytes is directed toward the perisinusoidal space and thus to the blood stream, in view of the close communication of this space with the sinusoids. The plasma membrane forms irregularly-shaped microvilli as a reflection of an extensive exchange between cell and environment, probably mainly involving ions and water. The microvilli extend to a varying degree between neighboring liver cells. In situations of stress, like extrahepatic biliary obstruction or cirrhosis, they are found deep in the spaces between the hepatocytes. This might be associated with excess water uptake (pinocytosis) and swelling of the cells. Contrariwise, the microvilli might disappear and the plasma membrane appear broken, with intracellular material in the perisinusoidal space. While some (15) consider this an intravital phenomenon, others believe it to be a fixation artifact facilitated by increased intravital fragility of the cell membrane. Both possibilities suggest an alteration of the cell membrane permeability. Possibly, this is clinically reflected in the increased serum activity of intracellular enzymes, specific for the hepatocytes or not, of which the transaminases represent the most widely used example (6). However, the elevated serum activities have also been explained by compartmental shift within the hepatocytes, i.e., changes of organelle permeability. Differences in hydration presumably indicating altered permeability are also reflected in the appearance of dark and light cells with varying content of intracellular fluid. This process may proceed to a complete dehydration of the hepatocytes as seen in the acidophilic bodies, most frequently found in viral hepatitis. They represent dense "mummies" with little intracellular fluid. Such cells, despite their morphologically-intact organelles, seem to be dead because they are attacked and devoured by surrounding macrophages (16).

The relatively small portion of the cell surface which lines the bile canaliculi between the liver cells is specifically modi-

fied by the formation of regular microvilli which fill the narrow lumen of the canaliculus. Both in extrahepatic biliary obstruction and in intrahepatic cholestasis, a characteristic alteration occurs which is the morphologic substrate of cholestasis. The latter is recognized by visible intrahepatic accumulations of bile pigment and functionally by elevation in serum of all biliary substances. This includes conjugated bilirubin and dyes, such as bromsulfalein, administered for test purposes, cholesterol, bile acids (responsible for itching) and alkaline phosphatase. The bile canaliculi are dilated to varying degrees. But the junctional complexes, which seal the canaliculi and thus prevent back flow of bile into the intercellular space, remain intact in cholestasis. The microvilli are either bleb-like and widened or more frequently stunted and absent (17). This is associated with a widening of a pericanalicular hyaloplasmic zone and with accumulation of electron opaque, presumably biliary, material in the vesicles of the Golgi apparatus. Cholestasis has thus been defined as a disturbance of the bile secretory apparatus of the hepatocytes, brought upon by pressure in extrahepatic biliary obstruction and by a primary toxic or metabolic alteration in intrahepatic cholestasis (6). In most clinical examples it is not, as previously had been believed, an alteration of ductules or cholangioles; the term "cholangiolitis", therefore, does not refer to the primary target of the injury in intrahepatic cholestasis. The fine structural alterations of the ductules, if present, seem to be a secondary manifestation as indicated by recent experimental observations, which may explain the mechanism of cholestasis (18). Lithocholic acid, a bile acid, foreign to mammals, but formed in the intestine by bacteria, interferes with the normal secretion of water moved by endogenous bile acids. This interference alters the canalicular microvilli and results in stagnation of biliary material, apparently in intracanalicular mucopolysaccharides acting as sponge-like resin.

Intrahepatic cholestasis can thus be considered as one type of hepatocytic injury which may be present alone as in early extrahepatic biliary obstruction or in some drug-induced hepatic injuries, as those produced by anabolic steroids, or even in rare cases of viral hepatitis. Usually it is combined, as in most cases of clinical liver disease with jaundice, with lesions of other organelle groups in the hepatocytes. By contrast, in anicteric viral hepatitis the injury to the bile secretory apparatus of the hepatocytes is missing.

This concept that cholestasis is one distinct type of hepatocytic injury not only has practical significance but also was a somewhat naive initial attempt at an organelle pathology. Two types of damage of the same cell are assumed with clearly defined differences in clinical and functional manifestations. At this time, one might recognize the dysfunction of one group of hepatocytic organelles concerned with the relation between the hepatocyte and the blood, be it resorptive or secretory, resulting in what is called hepatic insufficiency and involving the main functions of the organ. The other group of organelles concerns the hepatocyte-bile relation with the dysfunction representing a secretory defect. Various degrees of both hepatocytic dysfunctions account for the great variation of manifestations of hepatic disease which challenge the diagnostic astuteness of the clinician. Not all elevations of conjugated bilirubin imply cholestasis. In chronic idiopathic jaundice with pigment deposition (Dubin-Johnson Syndrome) or without it (Rotor Syndrome), the bile secretory apparatus is normal even on electron microscopy. Cholestasis is not seen, and only endogenous and exogenous pigments, but not other biliary substances, pile up in the serum. This seems to represent a defect in the

transport of these pigments in the hepatocytes, the morphologic substrate of which is today unknown.

The organelle response to injury is not only catabolic, but, just as in the response of whole cells and tissues to injury, other types of processes are initiated. One is repair of focal damage of the cytoplasm with preservation of the whole cell. Groups of damaged organelles are segregated from the rest of the cytoplasm. A membrane is formed around them and digestive enzymes accumulate. Acid phosphatase, because its reaction product is readily demonstrated by light and electron microscopy, is a favorite marker although its specific function in the digestive process is still problematic. The activity of this enzyme has served in the development of the lysosome concept of an intracellular digestive apparatus by de Duve (19). This concept describes accumulations of hydrolytic enzymes in organelles which are segregated portions of the cytoplasm not only for purposes of uptake, secretion and storage, but also for potential destruction of the cell or for local digestion of foci of cytoplasmic degradation (20). They become autophagic vacuoles in which the original organelles, like mitochondria or ER and even glycogen, can be initially recognized. Eventually, digestion is completed with the residue being oxidized unsaturated fatty acids which are pigmented and then form lipofuscin. Pigment deposition thus indicates preceding injury or aging of the cells.

Intracellular regeneration, in contrast to repair, has been recognized in new formation of both granular ER and of mitochondria. The former is reflected in the arrangement of polyribosomal helices in straight lines preceding the development of new ER profiles as seen both in the fetus (21) and in cirrhosis. Excess of mitochondria is seen, for instance, in prolonged hyperoxic circumstances (11). Little is known today as yet as to the messenger action which regulates intracellular organelle regeneration in a surviving cell.

A most challenging area yielding far-reaching speculations is the adaptation of the cell to stress. This can be a changed task produced, for instance, by an exogenous toxic agent, antibiotics or antimetabolites, or by an altered intracellular milieu as a result, e.g., of excess or decreased oxygen supply, or of an altered regulation by variation of hormones, particularly of the steroid variety. The phospholipid membranes of the ER are the sites of processing enzymes. Their prototypes are the drug-handling enzymes concerned with a variety of biochemical processes, such as hydroxylation, oxidation, methylation, dimethylation or nitroreduction. They have in common the transformation of preferentially lipid-soluble non-polar substances, aggregated in the phospholipid membranes, to polar water-soluble and thus easily excreted products, which means detoxification. Administration of a variety of drugs, exemplified by phenobarbital, chlorpromazine or pesticides, increases the smooth endoplasmic reticulum in experimental animals (22, 23) and man associated with accumulation of enzymes of the processing type. Since this is prevented by puromycin, it represents new synthesis. Enzyme induction is under control of steroid enzymes (24). The nature of the enzyme induced, however, seems to vary in that not only enzymes concerned with the substance or its metabolites are induced but also unrelated enzymes. After chlorpromazine or pesticide administration, the phenobarbital metabolism is accelerated, reflected in a shortened sleeping time. The pattern of the enzymes induced depends not only on the drug but also on cellular factors, such as hormonal regulation, and probably on previous exposure and on genetic characteristics. This is analogous to the serum gamma-globulin elevation following an immunologic stimulus which might include formation of specific antibodies and other, unrelated an-

tibodies. While enzyme induction precedes the increase in phospholipid synthesis, measured by isotope techniques, and the appearance of excess membranes, the latter may persist when the enzyme activity declines (25).

The smooth ER is also the site of hepatocellular cholesterol formation (26), and it is probably concerned also with processing of endogenous material since it shows focal proliferation also in human viral hepatitis or biliary obstruction. It also seems to preferentially handle water-soluble material since it proliferates also after alcohol intake (12).

This ER alteration may proceed to a peculiar whirl formation, which if excessive is even recognized with the light microscope as eosinophilic inclusions, e.g., in human viral hepatitis or in rodents after pesticide administration (27). The adaptive process is by itself initially beneficial, and its morphologic demonstration after drug administration cannot be utilized to demonstrate a no-effect level if a no-detrimental effect is meant. However, both from an academic standpoint and from a practical toxicologic one, the question arises where the border between beneficial and detrimental lies, i.e., what is a toxic response or an anabolic one. In other words, when does the functional or structural demonstration of the anabolic reaction recommend caution in the use of a drug? Long term administration of phenobarbital, a strongly anabolic agent, to animals has not been associated with hepatotoxic injury or shortened survival of the animals, though hepatitis in man after phenobarbital administration has been reported (28).

The new formation of the ER is only one parameter of the anabolic reaction, because it is, as a rule, associated with enlargement of the cell and of the whole liver. This means that the excess formation of the smooth ER is accompanied by new formation of other cytoplasmic elements. As a matter of fact, as already reported, other organelles like mitochondria may increase in adaptation, for instance, to hyperoxic conditions (29). Excess microbodies, the site of peroxidizing enzymes, may appear following alcoholic excess (12) and after exposure to some drugs (30). Enlargement of the cell and organ (hypertrophy) may occur even without new formation of processing enzymes or ER following prolonged exposure to Halothane (31). Protein synthesis may increase following exposure to drugs and sometimes even to toxic agents and it may thus not necessarily be reduced as result of potentially injurious stress. This has been found to be the case in cloudy swelling (32, 33) as predicted almost 40 years ago and in nutritional disturbances, such as in deficiencies of single essential amino acids (34, 35).

A biphasic reaction is thus suggested in that increase in protein synthesis might precede its depression, the latter associated with alteration of the rough ER. In the case of the pesticide, dieldrin, this biphasic reaction has been established in our laboratory (36). The administration of smaller amounts for shorter periods increases significantly the smooth ER parallel with an increase of aniline hydroxylase, as an example of a processing enzyme, and also the size of the liver. Larger doses or prolonged administration result in a reduction of the hepatic activity of aniline hydroxylase without significant decrease in smooth ER and in liver size. However, at this time, autophagic vacuoles and alterations of the mitochondria are seen and the hepatic activity of a mitochondrial enzyme, proline oxidase, is abnormally low. This implies that either intoxication of organelles other than the ER or mere stress upon them created by excess need for cofactors may damage the cell in the course of events starting with a beneficial adaptive induction. The portion of the biphasic curve of protein synthesis between the rising and declining limbs may thus

represent the border between adaptation and toxicity.

Enzyme induction may also be detrimental if one of the enzymes induced is by itself harmful. An example of damage to other organs from enzyme induction is the excess hepatic synthesis of Δ-amino- levulinic acid synthetase by various agents which produce prophyria (37). This is also associated with excess smooth ER as in the case of allylisapropylacetamide. The possibility also exists that excess enzyme induction results in hepatocellular injury particularly if proteolytic enzymes are induced. Such new synthesis of enzymes, together with their release from lysosomes, might be a mechanism to explain hepatic necrosis. So far this possibility has not been well demonstrated in experimental circumstances (38, 39), but it might represent the mechanism underlying the rapid hepatocellular digestion in "acute yellow atrophy". Increased proteolytic activity in this condition is fully established and it even continues after death, but whether increased synthesis of proteolytic enzymes explains the explosive destruction of the cells is a matter of future investigation. The hepatitic drug reactions with their known tendency to acute atrophy occur in an extremely low incidence in persons exposed to the drugs (40). They may possibly result from an excessive enzyme induction under specific genetic or immunologic circumstances. The somewhat more common cholestatic drug reactions may be related to another type of enzyme induction. These assumptions are supported by the fact that all drugs eliciting hepatic reactions are potent enzyme inducers (41).

The hypertrophy or enlargement of the single liver cell may or may not be accompanied by new formation of cells (hyperplasia). The latter may contribute to the enlargement of the organ. While some agents lead only to hypertrophy and others only to hyperplasia, some produce both. Moreover, with hypertrophy, hyperplasia might even be suppressed (42). Hyperplasia can develop without death of cells but is presumably stimulated by reduced function of the organelles of viable liver cells. The hyperplasia characteristically involving fat-free hepatocytes in hepatic steatosis (43) may thus be explained.

Hyperplasia is best demonstrated using radioisotope techniques, since the analysis of the nucleus in tissue sections by electron microscopy has lagged behind the study of the cytoplasm for technical reasons which are in the process of being overcome with modern fixation techniques. Understanding of the hyperplastic mechanism and the relation between hypertrophy and hyperplasia which varies in different organs (44) may represent a means of understanding uncontrolled hyperplasia and cancer.

The hepatocyte, despite its deceptively simple structure on conventional microscopy, has, under the electron microscope, an elaborate structure in keeping with its diversified functions. In response to stress, extensive organelle alterations frequently occur before hyperplasia sets in, and the life span of the hepatocyte is very long. By contrast, the mesenchymal cell has, as a rule, only one main function and differentiates mostly in one direction. This holds true also for the proliferation of the different hepatic mesenchymal cells which accompanies almost all types of hepatocellular injury and which is responsible for the name "hepatitis" applied to most diffuse liver injuries. Study of cell population kinetics indicates that the stimulated hepatic mesenchymal cell has a much shorter life span than the hepatocyte and, therefore, proliferates far more extensively than the static histologic section suggests (45). Hyperplasia of the inflammatory or mesenchymal cell is initiated far more rapidly following organelle alterations, which implies differentiation in one main direction. This is characteristic

not only for the hepatic macrophages or Kupffer cells but also for the fibroblasts and for the hepatic lymphoid cells, for which the rules of immunology in general apply, that differentiation is speedily associated with new cell formation. Whether death of hepatocytes or focal cytoplasmic degradation (autophagic vacuoles) is a stimulus for the hepatic mesenchymal cell reaction remains to be established.

Summarizing, then, we have emphasized the role of alterations of organelles in living hepatocytes and claimed that this is better related to the clinical manifestations of liver disease than death of cells. The correlation of functional and structural alterations of the organelles, following the path initiated by Palade (46), is probably one of the most promising aspects of pathology, at least of the liver. In closing, one might be led to speculate and to compare the intracellular evolution of the organelle alterations in response to injury with the well known evolution in whole cells. Change initiated by various stresses including toxic agents, alteration in cellular milieu or variation in hormonal regulatory factors may lead to an adaptive anabolic reaction which if not adequate in coping with the stress may in turn progress to catabolic, reparative and eventually regenerative organelle alterations. This might be followed by hyperplasia to meet persistent stress by augmenting the total number of organelles by addition of more cells to the organ. However, just as cellular adaptation in immunology or in non-immunologic inflammation may in turn damage the cell for the protection of which the inflammation was originally stimulated, organelle adaptation may be beneficial or dangerous, the latter in the sense of a feedback mechanism or vicious cycle. Since, notoriously, catabolic liver injury is a poor target of treatment, it might be therapeutically more promising to break this cycle at one of its other links, such as the anabolic phase.

REFERENCES

1. Hoppe-Seyler, G.: Beitrag zur Kenntnis der trueben Schwellung auf Grund chemischer Untersuchungen. Krankheitsforschung **6:** 323, 1928.
2. Leevy, C. M.: Fatty Liver: A study of 270 patients with biopsy proven fatty liver and a review of the literature. Medicine **41:** 249, 1962.
3. Popper, H., Steigmann, F., and Szanto, P. B.: Quantitative correlation of morphologic liver changes and clinical tests. Am. J. Clin. Path. **19:** 710, 1949.
4. Steiner, J. W., Phillips, M. J., and Miyai, K.: Ultrastructural and subcellular pathology of the liver. *International Review of Experimental Pathology*, Vol. III, p. 65. Edited by Richter and Epstein. Academic Press. New York & London, 1964.
5. Schaffner, F.: Intralobular changes in hepatocytes and the electron microscopic mesenchymal response in acute viral hepatitis. Medicine *45:* 547, 1966.
6. Popper, H., and Schaffner, F.: Fine structural changes of the liver. Ann. Intern. Med. **59:** 674, 1963.
7. Smuckler, E. A., and Barker, E. A.: Effects of drugs on amino acid incorporation in the liver. *Experimental Study of the Effects of Drugs on the Liver.* Proceedings of the European Society for the Study of Drug Toxicity **7:** 83, 1966. (International Congress Series No. 115, Excerpta Medica Foundation).
8. Novikoff, A. B., Roheim, P. S., and Quintana, N.: Changes in rat liver cells induced by orotic acid feeding. *Biochemical Pathology*, p. 27. Edited by Farber and Magee. Williams & Wilkins Co. Baltimore, 1966.
9. Baglio, C. M., and Farber, E.: Reversal by adenine of the ethionine-induced lipid accumulation in the endoplasmic reticulum of the rat liver. In Press.
10. Roheim, P. S., Switzer, S., Girard, A., and Eder, H. A.: Alterations of lipoprotein metabolism in orotic acid-induced fatty liver. *Biochemical Pathology*, p. 21. Edited by Farber and Magee. Williams & Wilkins Co. Baltimore, 1966.
11. Schaffner, F., and Felig, P.: Changes in hepatic structure in rats produced by breathing pure oxygen. J. Cell. Biol. **27:** 505, 1965.
12. Rubin, E. and Lieber, C. S.: Early fine structural changes in the human liver induced by alcohol. Gastroenterology *52:* 1, 1967.
13. Schaffner, F., Loebel, A., Weiner, H. A., and Barka, T.: Hepatocellular cytoplasmic

changes in acute alcoholic hepatitis. JAMA, **183:** 343, 1963.

14. Porta, E. A., Hartroft, W. S., and de la Iglesia, F.: Hepatic changes associated with chronic alcoholism in rats. Lab. Invest. **14:** 1437, 1965.
15. Rouiller, C., and Jezequel, A. M.: Electron microscopy of the liver. *The Liver: Morphology, Biochemistry, Physiology*, Vol. I, p. 195. Edited by C. Rouiller. Academic Press. New York & London, 1963.
16. Klion, F. M., and Schaffner, F.: The ultrastructure of acidophilic "Councilman-like" bodies in the liver. Am. J. Path. **48:** 755, 1966.
17. Schaffner, F., and Popper, H.: Morphologic studies of cholestasis. Gastroenterology **37:** 565, 1959.
18. Schaffner, F., and Javitt, N. R.: Morphologic changes in hamster liver during intrahepatic cholestasis induced by taurolithocholate. Lab. Invest. *15:* 1783, 1966.
19. de Duve, V.: General properties of lysosomes. *Lysosomes*, P. I. Ciba Foundation Symposium. Edited by Reuck and Cameron. Little, Brown & Co. Boston, 1963.
20. Hruban, Z., Spargo, B., Swift, H., Wissler, R., and Kleinfeld, R. G.: Focal cytoplasmic degradation. Amer. J. Path. **42:** 657, 1963.
21. Jezequel, A-M., Arakawa, K., and Steiner, J. W.: The fine structure of the normal, neonatal mouse liver. Lab. Invest. **14:** 1894, 1965.
22. Remmer, H.: Detoxification of drugs in the liver. *Progress in Liver Diseases*, Vol. II. Edited by Popper and Schaffner. Grune & Stratton. New York, 1965.
23. Fouts, J. R.: Factors influencing the metabolism of drugs in liver microsomes. Ann. N. Y. Acad. Sci. **104:** 875, 1963.
24. Conney, A. H., Schneidman, K., Jacobson, M., and Kuntzman, R.: Drug-induced changes in steroid metabolism. Ann. N. Y. Acad. Sci. **123:** 98, 1965.
25. Orrenius, S., and Ericsson, J. L. E.: Enzyme-membrane relationship in phenobarbital induction of synthesis of drug-metabolizing enzyme system and proliferation of endoplasmic membranes. J. Cell. Biol. **28:** 181, 1966.
26. Jones, A. L. and Fawcett, D. W.: Hypertrophy of the agranular endoplasmic reticulum in hamster liver induced by phenobarbital (with a review on the functions of this organelle in liver). J. Histochem. Cytochem. **14:** 215, 1966.
27. Ortega, P.: Light and electron microscopy of Dichlorodiphenyl-trichloroethane (DDT) poisoning in the rat liver. Lab. Invest. **15:** 657, 1966.
28. Klatskin, G.: Toxic and drug-induced hepatitis. *Diseases of the Liver*, 2nd Ed., p. 491. Edited by Schiff. J. B. Lippincott Co. Philadelphia, 1963.
29. Schaffner, F., Roberts, D. K., Ginn, F. L., and Ulvedal, F.: Electron microscopy of monkey liver after exposure of animals to pure oxygen atmosphere. Proc. Soc. Exp. Biol. Med. **121:** 1200, 1966.
30. Svoboda, D. J., and Azarboff, D. L.: Response of hepatic microbodies to a hypolipidemic agent, ethyl chlorophenoxyisobutyrate (CPIB). J. Cell. Biol. **30:** 442, 1966.
31. Kunz, W., Schaude, G., Schmid, W., and Siess, M.: Stimulation of liver growth by drugs. I. Morphological analysis. *Experimental Study of the Effects of Drugs on the Liver*. Proceedings of the European Society for the Study of Drug Toxicity **7:** 83, 1966. (International Congress Series No. 115, Excerpta Medica Foundation).
32. Popjak, F.: The mechanism of parenchymatous degeneration produced by diphtheria toxin. J. Path. Bact. **60:** 75, 1948.
33. Guidotti, G., and Melli, M. L.: Amino acid transport by the hepatic cell in cloudy swelling. Proc. Soc. Exp. Biol. Med. **110:** 719, 1962.
34. Sidransky, H., Staehelin, T., and Verney, E.: Protein synthesis enhanced in the livers of rats force-fed a threonine-devoid diet. Science **146:** 766, 1964.
35. Kean, E. A.: Effect of a methionine-deficient diet on amino acid incorporation in rat liver cell fractions. J. Nutr. **90:** 91, 1966.
36. Klion, F.: Dieldrin induced adaptive and toxic changes in the liver. Fed. Proc. **25:** 668, 1966.
37. Granick, S.: The induction *in vitro* of the synthesis of Δ-aminolevulinic acid synthetase in chemical porphyria: A response to certain drugs, sex hormones and foreign chemicals. J. Biol. Chem. **241:** 1359, 1966.
38. Slater, T. F.: Lysosomal changes in experimentally induced liver injury. Proc. Roy. Soc. Med. **59:** 877, 1966.
39. Nelson, B. D.: Hepatic lysosome and serum enzyme alterations in rats exposed to high altitude. Amer. J. Physiol. **211:** 651, 1966.
40. Popper, H., Rubin, E., Gardiol, D., Schaffner, F., and Paronetto, F.: Drug-induced liver disease. A penalty for progress. Arch. Intern. Med. **115:** 128, 1965.
41. Popper, H.: The problem of hepatotoxicity. *Experimental Study of the Effects of Drugs on the Liver*. Proceedings of the European Society for the Study of Drug Toxicity **7:** 9, 1966. (International Congress Series No. 115, Excerpta Medica Foundation.)

42. Barka, T., and Popper, H.: Hepatic enlargement and liver injury. Medicine. In Press.
43. Popper, H., and Elias, H.: Histogenesis of hepatic cirrhosis studied by the three-dimensional approach. Amer. J. Path. **31**: 405, 1955.
44. Gross, R. J.: Hypertrophy vs. Hyperplasia. Science **153**: 1615, 1966.
45. Rubin, E., Hutterer, F., Danon, G., and Popper, H.: Desoxyribonucleic acid turnover in a heterogeneous population—Life span of hepatic ductular cells. Lab. Invest. **12**: 657, 1963.
46. Palade, G. E.: Structure and function at the cellular level. JAMA, **198**: 143, 1966.

Endotoxin Shock[1]

By DONALD G. McKAY, M.D.[2]

OUR interest in endotoxin shock was stimulated some years ago by observations made at the time of autopsy on patients who had succumbed to disease processes caused by infection with gram-negative bacteria. Our first exposure to the problem came from cases of infected abortion and pregnant women at term with premature rupture of the membranes and chorioamnionitis (1, 2). We set ourselves to the task of attempting to reconstruct the mechanisms leading to death in these patients by examining the clinical and pathologic manifestations.

Clinically, the patients with infected abortion had had uterine bleeding followed by the gradual onset of fever for 2–3 days. They then had developed a severe hypotension which failed to respond to the usual therapeutic measures including the use of antibiotics, blood transfusions and plasma expanders, cortisone and vasomotor active drugs. Patients with term pregnancies who ruptured their membranes and then failed to go into labor also developed fever lasting 2–4 days. At this time the onset of labor was associated with the sudden appearance of hypotension and occasionally a severe hemorrhagic diathesis due to incoagulable blood. Aside from the few patients dying of massive hemorrhage, examination of the clinical data revealed that there was an association between a gram-negative infection, severe hypotension and hemorrhagic phenomena, but failed to explain the mechanism of either the hemorrhage or the shock.

Turning to the autopsy examination, a variable gross pathologic picture was found. In general, a variety of organs were involved in a process characterized by hemorrhage and necrosis. Specifically these included skin petechiae and ecchymoses, focal or complete hemorrhagic necrosis of the adrenal, petechiae or hemorrhagic ulcers of the mucosa of the gastrointestinal tract, small pulmonary hemorrhages, foci of necrosis of the anterior lobe of the pituitary and occasionally bilateral renal cortical necrosis. Aside from the few patients dying of uremia due to total destruction of the renal cortices, the gross autopsy examination only added the fact that irregular tissue necrosis was a common event and failed to provide evidence of the mechanism of the necrosis, the hemorrhagic diathesis or the shock.

Recourse to the light microscope was more enlightening. Many of these patients exhibited platelet and fibrin thrombi in the microcirculation. These thrombi were found in arterioles, capillaries, and venules of most of the viscera, in the same irregular distribution as the foci of hemorrhage and necrosis. From the histology it was clear that the foci of hemorrhage and necrosis were due to obstruction to the flow of blood in the various capillary and sinusoidal beds. More importantly, these capillary platelet and fibrin thrombi clearly proved that many of these patients had a process which in its simplest terms amounted to *disseminated intravascular coagulation*. Also, the development of incoagulable blood and massive hemorrhage in some of these patients could now

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be understood as due to the consumption of the components of the hemostatic mechanism. Hematologic studies (3) have shown that these patients have a sudden acquired depletion of circulating platelets, fibrinogen, Factor V, Factor VIII, prothrombin complex and Factor IX. This is due not only to consumption by the process of intravascular coagulation but to activation of plasminogen which in its active form digests fibrinogen and certain other components of the clotting system.

Very similar clinical and pathologic observations were made in patients dying from infantile diarrhea due to B. coli O-111-B_4 (4) and in patients dying from meningococcemia (5). Up to this point, clinical and pathologic examination had shown that there was an association of gram-negative bacterial infection with disseminated intravascular coagulation, shock, focal tissue necrosis and a hemorrhagic diathesis.

But there were important questions remaining. The major problem was what had caused the disseminated intravascular coagulation. For the answer it was necessary to turn to animal experiments.

Small doses of bacterial endotoxin, as little as 0.2 mgm., injected intravenously produced platelet and fibrin thrombi in the lungs, liver and spleen. A second injection 24 hours later resulted in an increase in the thrombi in these organs and the appearance of thrombi in a new organ, the kidney. At the end of 4 hours after the second injection, every glomerular capillary of both kidneys was completely occluded by fibrin thrombi. Those animals surviving for 8 hours or longer then developed infarct necrosis of the renal cortices, the hallmark of the generalized Shwartzman reaction (6). It was thus established that, when the blood stream is exposed to small doses of bacterial endotoxin, disseminated intravascular coagulation is one of the major consequences. The pathologic manifestations were virtually identical to those observed in patients dying of gram-negative infections. The alterations of the hemostatic mechanism of the circulating blood followed the same pattern observed in man. The importance of the clotting mechanism in the development of the Shwartzman reaction was underlined by the fact that anticoagulants, i.e., heparin (7) and dicoumarol (8), and the massive activation of fibrinolysis by streptokinase (9) would prevent the reaction.

Taking all the evidence together, it was clear that bacterial endotoxin produced the hemorrhagic diathesis and the focal tissue damage by causing disseminated intravascular coagulation. This still left the question of the mechanism of the hypotension and shock.

Since the small doses of endotoxin used in producing the Shwartzman reaction did not produce death in shock, we then used large doses in amounts of 2 to 4 mgm. per kilogram in single injections into rats. This produced a lethal effect in all the rats tested within a 5 hour period. Curiously, less than half of the rats dying after large intravenous doses of endotoxin exhibited histologic evidence of intravascular clotting, even though the endotoxin used was the same. However, even though thrombi did not appear histologically in all animals, all exhibited a sudden drop in the circulating platelets and fibrinogen levels and evidence of rapid activation of plasminogen. The possibility was thus raised that intravascular coagulation was occurring even though not demonstrable histologically. To test this possibility a group of rats were pretreated with a single large dose of heparin prior to injection of lethal doses of endotoxin. In this group the fibrinogen level did not drop and virtually all the animals survived (10). These studies in the rat indicate that endotoxin shock is not only associated with disseminated intravascular coagulation, but

that this is the mechanism of the lethal outcome in this animal.

However, our experience in the past with the clotting mechanism in laboratory animals has taught us that species differences may be so great that one cannot directly transpose observations from one species to another. In order to test the question of species differences, we turned to an animal closer to man on the phylogenetic scale, namely, the Rhesus monkey. The work of Kuida (11) on the monkey had already indicated a minor role of intravascular coagulation in the pathogenesis of endotoxin shock. Animals subjected to doses of bacterial endotoxin of 7–10 mgm. per kilogram failed to show any evidence of intravascular clotting by light microscopy and we have confirmed this observation with larger doses (15 mgm. per kilogram). Nevertheless, when the hemostatic mechanism was examined during endotoxin shock, we observed a drop in circulating platelets and fibrinogen levels and a rapid activation of plasminogen, suggesting that (as in the rat) there might be intravascular coagulation that was not visible by light microscopy.

Because of the higher resolving power and greater accuracy of the electron microscope, we turned to this instrument to answer the question. Rhesus monkeys given a single injection of 15 mgm./kilogram of bacterial endotoxin were sacrificed at 15 minutes, 1, 2, 3 and 4 hours after exposure. Sections were taken from the heart, lungs, liver, spleen, mucosa of the small bowel and kidney and examined in the electron microscope (12). In the first hour after exposure to endotoxin the pulmonary capillaries were jam-packed with polymorphonuclear leukocytes. Most of the leukocytes were intact but some underwent disintegration and the contents of cytoplasm as well as their nuclei were floating free in the plasma. This sequestration and disintegration of leukocytes in the pulmonary vasculature accounts for the immediate but transient leukopenia always observed when endotoxin gains access to the blood stream. Also, platelets were aggregated in the circulating blood, especially in the sinusoids of the spleen. Many of these were degranulated or were virtually totally destroyed. The alteration most pertinent to the discussion was the presence of fibrin strands in the lumina of the capillaries, arterioles and venules of every organ examined in all the animals. The fibrin strands were frequently located near collections of platelets. They gave the appearance of floating free in the plasma in all organs except the liver. Only in the sinusoids of the liver was there a packing of platelets and fibrin into occlusive thrombotic masses. These electron micrographs demonstrate that endotoxin shock in primates is accompanied by disseminated intravascular coagulation. They indicate that platelets are damaged and the fibrin strands are formed in the circulating blood. Why they do not clump together to produce occlusive thrombi (aside from the liver) remains to be demonstrated. It may be that the activation of the fibrinolytic system digests the fibrin rapidly and prevents the obstruction of capillaries. It may be that the release of fibrinogen or fibrin split products results in the formation of an abnormal fibrin strand that lacks the tensile strength to occlude blood vessels.

The occurrence of disseminated intravascular clotting in endotoxin shock does not mean that it is responsible for the shock. In four monkeys treated with large doses of heparin (70,000–90,000 units given intravenously during the first 4 hours) following intravenous injection of 15 mgm. per kilogram of bacterial endotoxin, there was no protection against the hypotensive or lethal effects of endotoxin. These experiments indicate that some other cardiovascular or vasomotor effects of bacterial endotoxin are responsible for

the shock and death when large doses of endotoxin enter the blood stream of monkeys.

In order to maintain perspective it is important to note that there is ample experimental evidence to indicate that under certain stated conditions disseminated intravascular coagulation does contribute to the development of hypotension. Years ago we demonstrated in the dog that pretreatment with heparin prevents the hypotensive action of amniotic fluid infused intravenously. Also, it was shown that heparin pretreatment lessened the hypotensive effect of the infusion of incompatible blood into the dog (13). Recently, we have demonstrated that infusion of thrombin intravenously into Rhesus monkeys produces hypotension (14). This is apparently a dose-related response since animals with low doses of thrombin developed a transient and reversible hypotension while those receiving large doses died rapidly with an abrupt "irreversible" fall in blood pressure. The fatal outcome is due to the cessation of blood flow in the lungs, in essence an acute cor pulmonale.

Since we are primarily concerned with the problem in man, let us turn our attention back to patients with endotoxemia. Studies of autopsies and of the hemostatic mechanism leave no doubt that disseminated intravascular coagulation is a frequent occurrence in endotoxin shock in man. Is there any reason to expect anticoagulant therapy (specifically heparin) to be of benefit to these patients? On the face of it all, animal experiments suggest that if heparin is administered at the right time and in the right dosage to patients with small amounts of endotoxin in the circulating blood it can be expected to prevent the capillary thrombi and focal tissue necrosis. This in itself would reduce the morbidity and mortality to some extent. However, the experience with monkeys exposed to large doses of endotoxin suggests that heparin treatment in man cannot be expected to prevent death in endotoxin shock. Unfortunately, there is no way to measure the amount of endotoxin to which individual patients have been exposed.

The actual evidence in man is just beginning to come in, but there are individual case reports that indicate that heparin may have benefited the patient (15). These cases are too few to establish the point at this time. In all likelihood, a careful "double blind" study will be needed to determine the precise role of disseminated intravascular coagulation in the morbidity and mortality from gram-negative bacterial endotoxemia in man.

Summary and Conclusions

1. Disseminated intravascular coagulation is frequently found at autopsy by light microscopy in patients dying from diseases with gram-negative bacteremia.
2. The appropriate intravenous injection of *small doses* of bacterial endotoxin produces an identical pathologic pattern in experimental animals. This is characterized as the generalized Schwartzman reaction.
3. *Large intravenous doses* of bacterial endotoxin result in shock and death, frequently with no light microscopic evidence of intravascular coagulation.
4. In the *rat,* anticoagulation with heparin prevents both the generalized Shwartzman reaction and death in endotoxin shock.
5. In *primates,* electron microscopic examination reveals disseminated intravascular clotting in endotoxin shock. It is likely that the simultaneous activation of plasminogen with rapid destruction of fibrin prevents the formation of occlusive thrombi.
6. Anticoagulation by heparin does not prevent death in endotoxin shock in primates. This indicates that the cardio-

vascular effects of lethal doses of endotoxin are of greater importance.

7. Nevertheless, disseminated intravascular coagulation induced in primates by intravenous infusion of thrombin may cause (depending on the dose):
   a. Rapid death in shock due to cor pulmonale, or
   b. A transient hypotension and defibrination.

   Disseminated intravascular coagulation is at least a contributory factor in the production of hypotension.
8. In *man,* anticoagulation by heparin in a few reported cases offers the hope that therapy may be effective in:
   a. Preventing focal tissue damage to kidney, adrenal, liver and pituitary, and
   b. Decreasing the mortality rate in these diseases.

REFERENCES

1. McKay, D. G., Merrill, S. J., Weiner, A. E., Hertig, A. T. and Reid, D. E.: The pathologic anatomy of eclampsia, bilateral renal cortical necrosis, pituitary necrosis, and other acute fatal complications of pregnancy, and its possible relationship to the generalized Shwartzman reaction. Amer. J. Obst. and Gynec. **66:** 507–539, 1953.
2. McKay, D. G., Jewett, J. F. and Reid, D. E. Endotoxin shock and the generalized Shwartzman reaction in pregnancy. Amer. J. Obst. and Gynec. **78:** 546–566, 1959.
3. Case history from the Staff Conference of the Sloane Hospital. Sloane Hospital Bulletin, Vol. 12, pp. 15–21, 1966.
4. McKay, D. G. and Wahle, G. H., Jr.: Epidemic gastroenteritis due to Escherichia coli 0-111-B_4. II. Pathologic anatomy (with special reference to the presence of the local and generalized Shwartzman phenomena). Arch. Path. **60:** 679–693, 1955.
5. Margaretten, W. and McAdams, A. J.: An appraisal of the fulminant meningococcemia with reference to the Shwartzman phenomenon. Amer. J. Med. **25:** 868–876, 1958.
6. McKay, D. G. and Shapiro, S. S.: Alterations in the blood coagulation system induced by bacterial endotoxin. I. In vivo (generalized Shwartzman reaction). J. Exper. Med. **107:** 353–367, 1958.
7. Good, R. A. and Thomas, L.: Studies on the generalized Shwartzman reaction. IV. Prevention of the local and generalized Shwartzman reactions with heparin. J. Exper. Med. **97:** 871–888, 1953.
8. Shapiro, S. S. and McKay, D. G.: The prevention of the generalized Shwartzman reaction with sodium warfarin. J. Exper. Med. **107:** 377–381, 1958.
9. Kliman, A. and McKay, D. G.: The prevention of the generalized Shwartzman reaction by fibrinolytic activity. Arch. Path. **66:** 715–719, 1958.
10. Margaretten, W., Phillips, L. L. and McKay, D. G.: The effect of heparin on endotoxin shock in the rat. (In press).
11. Kuida, H., Gilbert, R. P., Hinshaw, L. B., Brunson, J. C. and Visscher, M. B.: Species differences in effect of gram-negative endotoxin on circulation. Am. J. Physiol. **200:** 1197, 1961.
12. McKay, D. G., Csavossy, I. and Margaretten, W.: Electron microscope study of endotoxin shock in the monkey. (In press).
13. Hardaway, R. M. and McKay, D. G.: Disseminated intravascular coagulation: a cause of shock. Ann. Surg. **149:** 462–470, 1959.
14. Whitaker, A. and McKay, D. G.: The effect of intravenous thrombin infusion on the blood pressure and coagulation system of Rhesus monkey. (In press).
15. Allen, D. M.: Heparin therapy of purpura fulminans. Pediatrics **38:** 211–214, 1966.

Preventive Approach to Community Practice[1]

By GEORGE T. HARRELL, M.D.[2]

WE are living in a time of great social change. Recent Federal and State legislation may well accelerate changes in the organization of medical practice. Recent announcements to the press indicate that support at the Federal level may see a change in emphasis from research to service. Research will continue to be supported, but future emphasis may be more on applied and less on basic research. Both trends will lead to greater emphasis on delivery of medical care in the community and will permit a preventive approach.

Historical

In the past, life was short and perilous. The causes of disease were unknown. Endemic diseases, particularly malaria and enteric infections, killed large numbers of people, most of whom were children. Until very recent times, it is likely that over half the children ever born died before the age of five. In addition, epidemic diseases such as typhus periodically decimated the population.

Illness was cared for in the home by the physician who had few potent drugs with which to treat his patients. Accurate observations from a clinical point of view were made on the causes of illness. The Hebrews recognized epidemics of dysentery in army camps and correctly related the outbreaks to improper disposal of human excreta. Observations made on the habits of cats led to religious laws of burying excreta outside the confines of the camp. We now recognize the scientific basis for these regulations. The antibiotics secreted by soil micro-organisms kill the pathogenic bacteria. The Romans recognized the transmission of disease by water. The provision of adequate supplies of pure water by systems of aqueducts, some of which are still in use today, represents one of the first examples of the interest of government in public health and the prevention of community-wide diseases.

Hospitals developed at the time of the Crusades drew their name from one group, the Knights Hospitalers. The first hospitals were pest houses to quarantine those returning from the East with leprosy. Patients in mental hospitals were chained to isolate them from society. The infirm and chronically ill were kept in almshouses which were supported by government. The Church assumed responsibility for care of acute illnesses and the thread of hospital support by religious bodies persists to the present day. Aseptic surgery was developed in private or voluntary hospitals. The increasing cost and complexity of surgical procedures and other techniques for medical care have led, in this country, to virtual disappearance of proprietary hospitals and to the development of our voluntary system. Certain illnesses in which the life history is long have had costs for care which exceed the resources of individual families. In the case of tuberculosis, society developed separate hospitals to share the cost as well as to protect the group from transmission of the disease. Psychiatric hospitals were developed from a humanitarian point of view,

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largely through government action and support. The care of crippled children has been supported largely by private philanthropy. Few of these efforts were specifically directed toward the prevention of diseases in the community.

Long Range Trends

In this country, we have seen a rapid change from a rural to an urban mode of life. Almost within a single generation cities have tended to merge through their suburbs into the megalopolis. The population has expanded at an almost explosive rate with the growth chiefly in the age groups at the ends of the spectrum—the young and the elderly. In addition, the population is much more mobile than in former years so that the role of the family is changing. Rarely, now, does a household contain more than a single generation. The grandparents to help care for illness and to watch over children no longer live in the same house with the rest of the family. The movement of families has vastly increased so that nearly a fifth of the people have changed their residences in the community in the past five years, and others have changed the city of residence. Continuity of care with the planning of preventive measures becomes increasingly difficult for the family physician if, indeed, the family has one.

A change in the character of illness has occurred in the last thirty years. In the past, most people died of acute infections—pneumonia, tuberculosis, malaria and diphtheria. The control of epidemics of infectious diseases was thoroughly discussed in the Arthur Parker Hitchens Lecture VII by Hinman in 1962. Public health measures for the control of food and water supplies and the disposal of sewage have been very effective at the community level in preventing transmission of infectious agents. At the individual level, immunization, particularly of children, and the development of chemotherapy have further reduced the ravages of infections. The general increase in the standard of living with improved nutrition and housing contributes to the prevention of the former chief causes of death. Now people die with chronic illnesses, particularly cardiovascular problems of all types, including heart disease and stroke, and cancer. In the chronic illnesses, the natural history of the disease is probably life-long and much more importance must be attached to genetic factors. Accidents are also a major cause of death at all ages. Many accidents have behavioral causes which should be approached from the preventive point of view. The causes of disability in addition to the chronic illnesses are emotional problems which overlay existing organic disease or which reproduce the symptoms of disease as psychosomatic problems. These changes in the character and pattern of illness have led to a lengthening of the life span, which is now greater in women than in men. These alterations in the causes of death and loss of time at work will lead to an alteration in the approach to the teaching of medical students. Many of these problems lend themselves to a community preventive approach.

The explosive growth of research has led to a doubling of biomedical factual knowledge approximately every ten years. This rate of increase poses a problem for information storage and retrieval to libraries and practicing physicians. Research has led to the development of chemotherapy for both treatment and prevention of infectious diseases. Other drugs have been developed for the alteration of mood and the interruption of the natural history of mental illness. It should not be forgotten that half the hospital beds in this country still are occupied continuously by patients with mental illness though the average stay of the individual patient has been shortened by the develop-

ment of drugs. The alteration of disease patterns and the interruption of the natural history of disease is leading to changes in the patterns of delivery for medical care.

In the evolution of the patterns of care, the greatest change has occurred since World War II. The increase in knowledge has led to greater complexity in the procedures and skills used in the care of patients. This change is well illustrated in laboratory and x-ray procedures which are done especially in the study of diseases of the heart and lungs. As a result, less dependence has been placed by the public on the family physician practicing alone. The unit of society, however, is still the family and it is on this unit that the impact of disease is felt. Disease has economic, cultural and social implications as well as the purely biologic ones.

More and more, the profession is sharing responsibilities for distribution of knowledge and the availability of skills. This trend has led to a rise in the number of physicians in group practice. It has led to an increase in specialization which has been accelerated by public demand for specialized care. The complexity of procedures and the changes in patterns of care are leading to the development of more types and greater numbers of paramedical personnel who are being given increased responsibility for portions of care.

Increased demands for all types of medical care have been made by the public. The increase in complexity of procedures with the same relative number of physicians to population has resulted in fewer home visits with more care being given to ambulant patients in an office setting. Much greater use of hospitals, both for diagnostic procedures as well as for treatment, has resulted from public demand and has been stimulated by insurance and pre-payment plans. Health is now considered a right of all people. The public is not now satisfied with the cure of illness but expects prevention of it and maintenance of a state of health. Increased demands have led to an increasing role of government in the delivery of medical care. The recent legislation for Medicare and for regional medical programs with the possible future development of centers for care of stroke, heart disease, and cancer are examples of government response to public demand. How these new programs will implement community approaches to prevention of disease is yet to be determined.

Projections into the Future

Even though changes are occurring in the patterns for delivery of care, we can expect the individual doctor/patient relationship to continue. Greater focus should be placed on the family as a frame of reference, regardless of the special field of interest to which the individual physician may restrict his practice. More emphasis should be placed on understanding behavior both at the group and individual level. Greater attention must be paid to the inherent variability of all living things and especially of human beings, whether sick or well. More group practice can be anticipated. Some practice groups will be homogeneous with all physicians having a common interest in the type of disease problem seen. Other groups will have representation of many specialty types of practice. The use of the health-related professions, especially in the prevention of disease or disability, should increase.

In recent years, the number of laboratory procedures in relation to hospital beds has roughly doubled every seven to ten years. A continued increase not only in the number of laboratory procedures but in their complexity can be anticipated. Because of this trend, a greater capital outlay for laboratory space and equipment can be anticipated. The

increased volume will permit programs of quality control to insure that the data given to the physician for interpretation are dependable.

These trends will increase the focus on the hospital as the community center around which programs of medical care revolve. Particularly in areas with small community hospitals developed under the Hill-Burton program, the pattern has evolved of physicians' offices clustering around the new facility. In this fashion, the available equipment can be shared by many physicians and the cost of acquiring and operating it spread through the community. This same principle is implied in the programs for development of regional centers for the treatment of stroke, heart disease and cancer. The implications for community programs of early detection and prevention of disease are great.

More care of chronic illnesses which extend over months or years can be expected. Evolution of nursing homes into extended care facilities, as part of or in conjunction with community hospitals, will be necessary to meet this demand.

These trends will permit application of research techniques to analysis of need for patient care as opposed to demand and to development of more effective means of delivery. Such research should lead to improved health and recognition of the preventive measures which can be instituted in local communities. Information storage and retrieval can be done by mechanical techniques, particularly with the application of computers to the problem. These techniques will change medical libraries in character. Application of computer techniques to community hospitals will change the character of patient records and the methods for monitoring treatment of individual patients with acute illnesses. Indeed, regional centers for storage and rapid retrieval of information on individual patients would permit the physician who has been asked to assume responsibility for a family moving into a community to learn what has happened in the past and to trace the genetic and other factors which might affect his recommendations for care.

Medical Education

These long range trends will be reflected in a change in the character of medical education. Historically, education in medicine has been by the apprenticeship method. In the past, the first physicians were probably priests who made observations on natural phenomena and who could predict dramatic occurrences such as eclipses or other celestial events. The physician became surrounded with an aura of magic and was expected to produce miracles. This thread of religion in medicine has persisted to the present day.

In the training of a physician, the scholarship of older practitioners was written down on papyri, manuscripts or in printed books which were accumulated in libraries. Teaching of techniques and skills required access to patients.

Medicine is now recognized as a true university discipline. The growth of science in medieval universities began in the faculties of medicine. The natural sciences of biology, chemistry and physics as we know them today evolved from efforts of physicians to improve their understanding of disease and methods of treatment of their patients. The laboratory approach to teaching and the inclusion of research techniques became firmly incorporated in the educational program as universities assumed more responsibility for medical education. Research will serve as the scientific basis for development of preventive programs. Better understanding of social and behavioral aspects of the community also will serve as the basis for development of community preventive programs in accidents, func-

tional overlay to disease, and mental illness.

For individual patient care, understanding of acute illnesses will continue to be taught in teaching hospitals. The frame of reference for understanding of reaction to stress and illness will become increasingly the family unit living in the community. For the community itself, public health measures as we know them will continue for the protection of society as a whole. Increasingly, the extension of preventive measures at the individual level can be expected. Great attention is given these days to prevention of environmental contamination of air and water supplies, particularly by industrial pollutants. Better understanding of population dynamics with recognition of genetic factors and understanding of fertility control will be essential for survival of many nations.

In the new Pennsylvania State University College of Medicine, a Department of Family and Community Medicine has been organized to improve the education of students. A Department of Behavioral Science has been organized to take a basic biologic approach as a medical science to understanding of group and individual reactions. The range of variation of the normal and of reaction to stresses of living as well as of illness will form the basis for understanding psychosomatic problems in all clinical disciplines as well as mental illness. Alterations in behavior are the basis for the functional overlay of existing organic disease and for understanding of psychosomatic illnesses. The prevention of accidents, a leading cause of death at all ages and especially in the young, should result from a better understanding of behavior. A Department of Humanities is being organized. Physicians must continue their education not only in the biological sciences but in the humanities as our culture evolves. It becomes increasingly important for the physician to recognize the sense of values of people and how they lead their lives. Unless he recognizes these intangible factors, acceptance of his recommendations by the family may block the delivery of optimum patient care.

Chronic Illness

A community approach to control of chronic illness should be planned. Screening techniques have been developed for detection of incipient chronic disease. These techniques utilize laboratory facilities and are frequently based on chemical methods. Dependable techniques for screening of incipient behavioral disturbances should be developed. All of these techniques will take manpower and facilities both in hospitals and in other settings. When chronic illness can be detected early, the possibility exists for the prevention of the complications of the disease process. The Department of Family and Community Medicine should offer an effective avenue for transmission of knowledge to the family about chronic illnesses and their courses, about genetic background and about the means all members of the family should adopt for prevention of complications.

Improved techniques for education of the patient and the family in the meaning of illness and the rationale behind the measures recommended for long term care should be explored. It may be necessary to develop new training programs for people who may serve as family health advisors. Study needs to be given to whether basic training as a nurse, with additional emphasis on health education and patterns of work with the individual physician, will be effective, rather than the institutional approach through the Public Health Department now used. The rehabilitation of patients after an acute episode of chronic illness will increasingly use the associated health professions and other paramedical personnel. All community resources will be

needed, whether supported by voluntary health agencies or government.

Conclusions

The doctor is faced, as he has always been, with a dilemma. The inherent biologic variability of all living things requires that a statistical approach be made in medical research to meet scientific criteria. Research increasingly is developing a scientific base for the practice of medicine.

The individual patient, however, does not wish to be treated as a statistic. Indeed, the application of data collected on an individual when interpreted in the light of conclusions drawn from studies of a group does not meet scientific criteria. The practice of medicine as it applies to the individual patient will remain, as it always has been, an art, even though it rests on a scientific base. The individual patient expects to be treated in a highly confidential interpersonal relationship. This approach will apply to his care the lessons learned from the study of the humanities. The dilemma comes when the physician is expected simultaneously to be a cool, objective, dispassionate scientist analyzing data and a warm, compassionate human being interested in another. A balance must be achieved between these two points of view.

A community approach to medical care can be developed with an emphasis on prevention which will lead to greater health of all the people in the future.

Notes on a Summer in Vietnam[1]

By DAVID J. McLANAHAN, A.B.[2]

THE following is an account of some of my experiences in Vietnam during the two months I spent there in the summer of 1966. I was one of five American medical students sponsored by the State Department's Agency for International Development. We each worked in a different civilian hospital at which AID has an American Advisory Team. I was at Danang.

I was fortunate in being able to get a good view of U. S. operations in Vietnam, policies in action at the local level, the general situation in South Vietnam, and the reaction of some Vietnamese people to all of these. I went through a series of briefing sessions which AID gives its long-term personnel upon arrival in Saigon; I was able to travel around some of the northern part of South Vietnam, and I had a close relationship with six Vietnamese medical students and a young ARVN surgeon.

My purposes in going to Vietnam were to provide medical care for civilian war casualties and to learn as much about the general situation as I could while fulfilling this function. AID's purpose for us, according to the Chief of AID Public Health in Vietnam, was to prevent the spread of communism in Asia. Our part in this effort would be to help win the peasant to the government side, in part by showing him that the government could take care of his health needs. Other considerations were the humanitarian aspects of relieving suffering and enhancing our own education in tropical medicine and medical problems in underdeveloped countries. Another hope, presumably, was to make us defenders of the U. S. position in Vietnam upon our return home.

My first patient was a lovely 28-year-old peasant woman who was lying on her back nursing a young child. The evening before she had been sitting in her thatched hut when a piece of shrapnel tore through her back, transecting the spinal cord. She was completely paralyzed below the nipple line. We could do nothing more for her than administer antibiotics and find a place for her to lie. A few mornings later she was dead and her body was carried back to her hamlet by relatives. This was a particularly poignant case, but typical of the tragedy seen daily in our emergency room and, most likely, all of the emergency rooms in Vietnam.

Ninety per cent of our patients had war-related traumatic injuries. Another five per cent were pedestrians or bicyclists struck by fast-moving military vehicles. Most of the patients were peasants brought in from the countryside by military trucks. Rarely did we get these patients less than sixteen hours after injury. All transportation ceases after dark. A small percentage of war casualties are lucky enough to make it to the hospital. I believe the figure is one in eight.

The Agency for International Development operates about forty civilian hospitals—the goal is to have one in each of the 43 provinces of South Vietnam. These are supposed to be in the province's area of major population concentration and to serve as a facility for more definitive treatment to which the outlying district clinics funnel patients. One of the AID supported hospitals (Danang) has a U. S. Public Health Service team, ten or twelve have "Free World Assistance" teams, and the

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FIG. 1. The author with a Vietnamese mother and her sick child

rest are staffed by U. S. military teams "on loan" to AID (MILPHAP or Military Provincial Health Assistance Program). About 100 physicians are involved in AID programs. There are 2–300 non-military Vietnamese physicians with Western-type training. The first line of defense for most of South Vietnam's 16 million people is traditional Sino-Vietnamese folk medicine.

Danang's Surgical Hospital (separated by a few blocks from the older Medical Hospital which is not U. S.-supported) has approximately 350 beds and at least two patients per bed. The pediatrics room often has four children per bed. There are always at least twice as many non-patients as patients. They sleep everywhere. The main cement-block building is a few years old. A two-story ward wing was opened earlier in the year, which has two rooms with pock-marked walls where government planes had strafed during the spring Buddhist crisis while aiming at a nearby radio station.

There are two wards of wooden lattice construction set apart from the main complex for burn patients. Functionally, they are open air—the Vietnamese do amazingly well with extensive body burns.

The *Médecin Chef*, or Vietnamese surgeon in charge of the Danang hospitals, is the much respected Dr. Tung. He had served two years of residency in obstetrics at Johns Hopkins University. The Americans are under his authority nominally, although they perform 95-plus per cent of the surgery. He is at the Surgical Hospital for an hour every day, making rounds and handling administrative matters. Dr. Tung has the reputation of being the best doctor in the northern part of South Vietnam. It is rare that other Vietnamese physicians come to the hospital. Generally, the relations between the Americans and Vietnamese are excellent.

The VN government pays all the Vietnamese hospital personnel and also provides them with room and board. The nurses receive nine or ten dollars per month. The patients are provided with everything required, including an adequate amount of food.

The logistical support is adequate. We never ran out of IV fluids or the important antibiotics. We could usually find some kind of drug for most occasions. Our situation was much better than the Medical Hospital which often ran out of antibi-

ones. Digitalis, etc. One of the other U. S. medical students, who was in charge of a pediatrics ward in Quang Ngai, sixty miles to the south, reported that they occasionally had to use coconut milk IV's—which is a common practice in outlying areas and of the Vietcong. We received ample outdated blood from the military hospitals and had good luck with it. Most of the other hospitals are chronically short of blood for transfusion.

The laboratory could do most of the simple tests—hematocrits, urinalyses, serologies, bacterial cultures and sensitivities. The old X-ray machine (a new one was being installed when I left) was adequate for most of the studies necessary in evaluating trauma.

Power failure was common and occasionally a real problem. Once it was responsible for the death of a girl in the operating room when suction failure allowed her to aspirate stomach contents.

The Vietnamese nurses change dressings, start (and sometimes order) IV's, and do other basic nursing chores. Most patient care is handled by the family of the patient. The surgical and emergency room nurses are quite good at suturing lacerations—even dirty ones—as they did most of this work in the days before the Americans arrived. There are twelve interpreters, who do a fairly good job. Some of the nurses can speak a little English.

About two-thirds of all AID-sponsored surgery in South Vietnam is or was done in Danang's two operating rooms. This amounted to 300 major and a far greater number of minor cases each month. At the beginning of the summer this was being done by two two-month volunteer surgeons and one long-term orthopedist, who I am sure is doing more traumatic orthopedic work than anyone in the world. When I left, there were three two-month volunteers and five long-term Public Health Service physicians included in the U. S. Surgical Advisory Team #1 at Danang. The new surgical suite of four operating rooms was opened a few days after I left. The aim of AID and Dr. Harry Wise, the new Medical Officer-in-Charge (U. S. Public Health Service), is to make Danang into a model hospital, practicing as close to American medical standards as the paramedical and logistical support will permit. The plan, which is an excellent one, is to make Danang into a training center for medical, nursing and technical students. Dr. Wise hopes to have eight Vietnamese medical students there at all times, to have Americans assist them in operations whenever feasible and to have Americans give some evening teaching lectures. Vietnamese students must go into the Army immediately after graduating, where, without specialty training, they often will be required to perform special procedures. There were two last-year students from the University of Saigon Medical School at Danang most of the time that I was there who, unfortunately, went back to Saigon before Dr. Wise arrived. In Saigon, each is in charge of a whole ward of patients for whom they do most of the surgery, including bowel resections, etc., and who came to Danang to find out how Americans do certain procedures, but they never found out as they spent most of their time sewing lacerations in the emergency room and taking care of ward duties. Before the arrival of Dr. Wise there was a polarization in attitudes among the Americans. One view held that we were there to do as much surgery as possible, without concern for teaching or working with Vietnamese students, nurses and anesthetists. The other (an official AID policy) view was that we were there to teach and work with the Vietnamese in order to prepare them to take over for themselves "when we leave". This situation should be much better under the capable and imaginative leadership of Dr. Wise.

The first few weeks I was in Danang I

spent most of my time as first assistant in the operating room and made rounds by myself, with one of the surgeons or with Dr. Tung. When more surgeons arrived, I worked more in the emergency room. I did more medical procedures in two months than I did in the past two years, more than I will have a chance to do in the next two years: cutdowns, thoracenteses, chest tubes, spinal anesthesia, lumbar punctures, castings, minor amputations, debridements, foreign body removals, major laceration repairs and an appendectomy. I also saw many unusual patients and pathology which my classmates will probably never see, such as huge head and neck tumors, typhoid bowel perforations, plague bubo, tetanus risus sardonicus, and many kinds of trauma, including that of a five-month pregnant woman with a bullet in her uterine cavity and of two women who had been shot through prolapsed uteri and nowhere else.

One of the most useful things I gained from the summer in Danang was experience in keeping calm in the face of anxiety—provoking life and death situations—such as the two hours I spent in the emergency room one weekend afternoon with no Americans or interpreter and with an avalanche of emergency patients needing immediate attention and operations. I will most likely lose my hard-earned knot-tying ability before I will really need it again, but will undoubtedly be in situations calling for tight self-control during my fourth year of medical school at Temple.

A valuable asset at Danang was the opportunity to work, talk and socialize with Vietnamese medical students. I had a close relationship with four students from Hue and two from the University of Saigon Medical School. We spent many evenings and weekends together—talking about medicine, politics and philosophy, and swimming, climbing, sightseeing, singing and playing guitars. The Vietnamese students I met could make it in any American medical school. They were being taught and knew the same things that I have been taught about Conn's Syndrome, for instance. They know about electrolytes, even though they rarely have a chance to use this knowledge. They use many of the same medical books I have—one Hue student was even translating Nelson's *Pediatrics* into Vietnamese. The Vietnamese students have a certain advantage over U. S. students in that their pathology/student ratio is much higher and they must learn to make firm clinical decisions earlier in their training than we do.

FIG. 2. Examining an injured patient in the emergency room at Danang Surgical Hospital. The author (center) with two Vietnamese medical students: Nghiem Dao Dai, University of Saigon (right); Tran-Viet Phon, University of Hue (left).

Although the patients did not talk freely with Americans about the sources of their injuries, it was possible (through interpreters and Vietnamese students) to estimate that 80 per cent of the war-related

trauma was due to Vietnamese government or United States military activities. Many of our patients were from "Vietcong families". Some were visited by intelligence officials. The *Médecin Chef* said that he received lists of names, villages and ages of suspected Vietcong in the hospital. There was no discrimination shown, however, in the treatment of patients.

A short description of a few of the cases met during my first hospital day (July 4th) and some of the other patients of the summer may make the situation appear more immediate. At 8:15 a.m., a truckload of casualties arrived, mostly mortar fire victims.

I helped a Vietnamese medical student debride shrapnel wounds of the back in a woman who also had a bad compound fracture of the base of the skull. A two-year-old boy who had been operated upon for an abdominal wound was very feverish and in an opisthotonic position. I did a spinal tap; the results were negative. It is likely he was too weak to hold up his head.

A boy with a traumatic A-V fistula of several days duration arrived in the emergency room, but shortly died of exsanguination. In the afternoon I helped at surgery with a woman who had stepped on a mine, which had blown off the front of one foot and the back of another. A boy came in at 4:30 p.m. with part of his hand missing. While holding a shell, trying to remove the powder, it had exploded. Other typical cases were a man with abdominal worm pain, a girl with *mittleschmerz*, and a three-year-old boy with a hydrocele. The most striking impressions of that first day were the huge patient load, the patients with multiple traumata and large tissue defects, and the almost ubiquitous post-operative wound infections.

During the summer a Navy jet crashed shortly after takeoff from the Danang airbase. It hit a populated area. A 1000-lb. bomb exploded, 40 mm. cannon bullets began firing and the ignited jet fuel created a very hot fire. Thirty-plus persons were killed and more than fifty injured. We got ten or twelve casualties at our hospital, most of them severely burned. Six were children, all about the same size and with first and second degree burns over most of their bodies. One nine-year-old boy was unforgettable, as both of his arms were seared completely dry and could have been snapped like pieces of wood. The same was true for his face, the eyelids parched shut, the lips and most of the nose gone. The mouth was frozen open; he breathed noisily through froth. Luckily, he never regained consciousness.

One morning we received two patients who had been injured four days previously. The man had a shrapnel wound of the thigh into which water buffalo dung had been stuffed to stop the bleeding. The leg was foul-smelling and greatly swollen. It had to be amputated. The other patient was a woman whose arm had been blown off just below the elbow. She had an edematous necrotic stump with fragmented radius and ulna poking out.

One afternoon we had nine os calcis fractures in six patients. They had been riding in a bus which ran over a mine. The buses which travel between provincial towns have floors lined with sandbags; these absorb most of the force of the explosion but also direct a component to the feet and legs of those standing on them.

One day we saw five cases of foreign bodies in the head, including three intracranial wounds. One was a girl shot in the back of the head who had a missile in an occipital lobe of her brain. Several square inches of brain tissue were exposed. She had visual changes in one eye and was sent to the Army neurosurgeon for care.

A ten-year-old girl had a wound of the abdomen which penetrated the stomach. She had a gastric fistula which would not heal. Everything she swallowed came out through it. Her laporatomy wound broke down and subsequent attempts to close it were without success as her tissues were

FIG. 3. Caring for a civilian casualty with the aid of Miss Martha Dickens, R.N., U.S.P.H.S., and a Chinese interpreter, Mr. Quang.

so friable. Eventually, the whole front of her abdomen became necrotic. She never complained. She was moved to the Marine Hospital where she could receive better nursing care.

The emergency room was a strangely quiet place. Very few of the patients gave more than facial reaction to pain. Patients with protruding compound fractures and traumatic amputations would just wrinkle their faces and cluck to themselves. Even most of the children were quiet. It was a privilege to just observe these heroic people. Women with severe injuries would nurse their infants while lying on the floor, in hallways or in the crowded wards. Every patient had at least one relative to take care of him. I once saw a crutches race across the hospital yard among three boys with amputated legs.

The severely injured would be fanned all day and all night and even after death. The Vietnamese peasant's handling of death is different from ours. Often there is an inability to recognize or comprehend death. Another American medical student was brought a dead girl to examine. The relatives had kept the body warm for three hours by charcoal heat. After the acceptance of death there is an emotional acting-out which lasts for a few minutes to a half hour. The mourner crys and screams and may roll on the floor. This outburst then suddenly stops and the dead body is carried back to the village.

The medical battle in South Vietnam is

a fierce one. Heavy casualties and treatment problems associated with the disruption of communications and transportation are increasing. Casualties are increasing at a greater rate than are medical facilities. The Danang Surgical Hospital represents the best in facilities and dedicated personnel available to civilians. Yet even Danang is little more than a station for emergency treatment, being so inundated with life-and-death situations that follow-up procedures are left undone. Patients go back to their villages with severe burn contractures, tissue defects and loss of function which could receive great benefit from follow-up plastic procedures. The chronically diseased must be sent home, often to die without medication, because of space and supply restrictions. The United States and AID have taken action to alleviate the situation. But the expenditure is woefully small in proportion to the effort which eventuates in the production of casualties.

Sometimes political strategy conflicts with good medical assistance. An example is the harassment of the medical school and hospital in Hue by the Saigon government and the lack of American financial aid or personnel to this 1500-bed medical facility and training center. The United States and AID, on A.M.A. advice, concentrate money and technical assistance in the University of Saigon Medical School (500 students) to the exclusion of that in Hue (200 students). This seems to me to be, in the long run, a self-defeating policy which is both alienating some of the future leaders of the country and preventing the emergence of a potentially fine medical center in an area of the country where the need is very great.

It is my opinion that the successful outcome of any plan to improve Vietnam's medical situation must depend upon a reduction in civilian casualties. To me, the steps to this reduction are obvious and of paramount importance. The overwhelming impact of the suffering of these innocent and graceful people is something that cannot be expressed by words.

Memoir of Charles C. Wolferth 1887–1965*

By FRANCIS C. WOOD, M.D.

DR. CHARLES C. WOLFERTH was elected a fellow of this College on June 8, 1918 and continued his association with us for over 47 years, until his death on December 26, 1965, when he was 78 years old.

He was my parent in medicine, and no parent was more admired and appreciated by his son, increasingly over the years.

Two excellent obituary notices have been written about him by Dr. T. Grier Miller (1) and Dr. Calvin F. Kay (2) which you may consult for a complete account of his life. I plan to omit some of the details and focus on certain elements which impressed me, and some things not so commonly known, which would not appear in a conventional *curriculum vitae*.

Dr. Wolferth had a number of remarkable qualities and made many important contributions to medical science. Yet, when I compare him to other great men I have known in medicine, the most unusual thing about him was that he possessed outstanding ability in both of two great areas of medicine.

First, he was a profound, penetrating, original, logical thinker and cool-headed master of the experimental method of medical science.

Second, he was one of the best and greatest physicians and consultants I have ever had the opportunity to know at close range, with all this implies in human understanding and consideration.

A number of men in my acquaintance have been superb in one of these areas, yet only "good" to "fair" in the other. Dr. Wolferth was truly outstanding in both. This remarkable binocular perspective enabled him to see the relationship of Clinical Science to Clinical Medicine with unusual clarity. His editorial in *Circulation* (3) is the best consideration of this relationship I have read. He was the epitome of the Clinical Scientist, whom he described in this editorial.

Charles Wolferth was born on May 18, 1887 at Wolferth Station (named for his grandfather) near Clarksboro, New Jersey. His father was a farmer, much interested in agricultural research. Charlie worked on the farm during his early school years. He went to Princeton (class of 1908) where he was a Phi Beta Kappa and a member of the football and swimming teams. He came to the medical school of the University of Pennsylvania (Class of 1912) where he was elected to AOA and to the presidency of his class. He played varsity football as a medical student. In the autumn of his fourth year, he did not report for football. A few days later Dr. J. William White, an ardent football fan, called Charlie to his office and asked him why. Charlie told him, "I am a poor boy. My future in medicine depends on my own efforts. I have had to slight my studies to play football. I have got to go to work and do better this year, because I want a good internship." Dr. J. William White asked, "Where do you want to intern?" Charlie said, "Here at the Hospital of the University of Pennsylvania." White, the professor of surgery, said, "All right, that's easy. You play football this year, and I will see to it that you get the internship you want."

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Dr. Wolferth went to France in the First World War and became a Major, Chief of the Medical Service of Base Hospital #31 at Contrexéville. He also had an opportunity to spend some time with Sir Thomas Lewis in England. When he returned to the University of Pennsylvania he had the opportunity to set up a Heart Station for the study and the treatment of patients with cardiovascular disease, patterned to some extent after Sir Thomas Lewis' model. Subsequently, Dr. William D. Stroud and his brother induced Mr. Edward B. Robinette to finance this operation. The Robinette Foundation was the result. Good scientific work is not measured by volume, but the reprints of the Robinette Foundation during Dr. Wolferth's directorship are contained in seven volumes and they are worthy of careful study.

Dr. Wolferth was the director of this foundation from its inception to 1952, when he became emeritus. He was the real genius of this group. Practically all the ideas which came out of it were his, or were added to significantly by him. He read and criticized every paper with care. I wish every young man in medicine, writing his first paper, could have the experience I had. Dr. Wolferth made me rewrite it 13 times to remove all unnecessary words, and to clarify all equivocal statements. He insisted that I read carefully each reference I wished to cite.

He never allowed his name to be put on a paper, unless he had made a definite contribution, and never permitted any order of authors except that indicating the true relative contributions of each. His mind was always out ahead of the experiment and ahead of everyone else. I might cite an example. One day I came back from the dog laboratory after some coronary artery experiments and told him that the anterior wall of the heart became blue when I clamped the left anterior descending coronary artery, but the limb leads of the electrocardiogram (which were all we were taking in those days) showed no change. Yet, an electrode right on this blue area on the front of the heart recorded a dramatic change.

The next morning he called me to his office and said, "I have been thinking about what you told me last night. You know we can't put an electrode on a patient's heart, but the next time a patient comes in with the clinical picture of coronary occlusion with no electrocardiographic evidence of it, why don't we put an electrode on the front of the chest, over the heart, and see what happens." And so, precordial leads were born with my muscle and his brain.

He made so many important contributions to the study and treatment of cardiovascular disease that I would not have time to mention even the significant ones. He studied all the various heart sounds more thoroughly and productively than any one of his time. His idea of explaining the Wolff-Parkinson-White Syndrome on an extra pathway of A-V conduction was characteristic of his mental processes. His advocating adrenalectomy for hypertension was an important step in our present understanding of many features of this disease. His study of the mitral valve at necropsy in patients with mitral stenosis led him to suggest valvulotomy years before it was actually done. He suggested plication of the enlarged left atrium in certain patients with mitral valve disease.

He rarely read editorial opinions. He wanted facts and formed his own independent opinions. His favorite extracurricular reading was the Wall Street Journal, although I am told he was an avid reader of philosophy in his earlier years.

He was a delightfully tolerant physician who viewed the foibles of his patients, his fellow physicians and himself with affectionate amusement. He used to say,

"I like my patients to have lots of bad habits, then their prognosis is better." He once observed, after hearing a flattering statement about himself, "It is amazing! An otherwise intelligent, critical person will believe absolutely anything about himself, no matter how fantastic, provided it is complimentary."

Yet, there was one subject on which he was not tolerant. If anyone mentioned "unipolar" electrocardiographic leads in his presence, you could almost see him take one look at the matador, snort, lower his head and charge. To the end of his life he used C-R precordial leads, never V leads. When someone would call the "unipolar lead" a useful approximation to the truth, he felt it was analagous to the "useful approximation" of Einthoven's equilateral triangle, which, in his opinion, held back electrocardiographic progress for years. Approximations, loose statements and loose thinking were an anathema to him.

Now, I should like to look at his capacities in the second of the two areas in which he was so outstanding—that of physician and consultant, his human understanding and consideration. One day I was in a quandary as to what to do about the management of a patient and consulted him. He thought for a while, then said, "Fran, that man is your father; now what are you going to do?" He was the best of consultants, especially in the cardiovascular field, but not limited to it. He objected to being called a cardiologist. He properly preferred to be known as an internist. He was the doctor's doctor. He continued in active practice throughout his medical life, and made rounds on his patients in the hospital on Christmas Day, 1965, the day of his fatal attack of dissecting aneurysm of the aorta.

The best account of this side of his life was written by Dr. William U. McClenahan. I quote the following with his permission:

"As a former student, and one who saw him in action as a consultant on many occasions, I should like to mention certain talents of special value in dealing with a conscious patient faced with a cardiac complaint, where fear and apprehension are manifestly ever-present complications. The proper management of such a patient requires more than a mere diagnosis and proper therapy. Dr. Wolferth showed an interest and understanding in the referring physician as well as in his patient. His prompt response and eagerness to help were always appreciated by both parties. A keen, dry sense of humor, always beautifully timed, was a real tonic. How few of us know how, when, or where to be funny! He listened longer and talked less than the average consultant. His brevity of speech was a simplication of the problem, and not an undue exaggeration of the picture so frequently noted in modern medicine. At the same time you never felt he was minimizing the seriousness of the situation. Slow, patient, unhurried thought, study and examination always preceded his opinion, even though to the less experienced the diagnosis and treatment seemed so obvious. Associated with an amazing ability to get to the real core of the problem faster, by far, than the average physician, was a flexibility of approach and management. Many of us through ignorance or fear seek the shelter of convention and routine. We sell ready-made suits of therapy to our patients. I never left a consultation with Charlie Wolferth without a sense of satisfaction, shared by my patient, that an individual tailor-made solution was offered to his particular specifications."

In closing, I should like to mention his outstanding characteristic, rare indeed in all masters, difficult to possess and hard to portray, but of such dynamic force—genuine humility associated with extreme confidence, two apparently contrary

forces blended together into one. This idea is so well expressed in his own words at the end of his biography in the Princeton class book, celebrating the 15th anniversary of his graduation:

> The annals of the poor, I understand, are brief. During all this time, nothing that could be of particular interest to the Class has happened to me. To the best of my knowledge, I have never been a hero, but on the other hand have managed, thus far, to keep out of jail.

To me, and to many who came under his influence, he *was* a "Hero"—a great example of what a physician might become if he had the necessary character, intellect and understanding. Something was once said about Dr. William B. Castle, which applies equally to Charles Wolferth —"to him that hath knowledge, wisdom and compassion will be given honor, even in his own country".

REFERENCES

1. Miller, T. G.: Charles C. Wolferth. Transactions of the Assoc. of Amer. Physicians **79:** 81–83, 1966.
2. Kay, C. F.: Charles C. Wolferth. Amer. Heart Journal **72:** 144–146, 1966.
3. Wolferth, C. C.: Clinical science, clinical medicine and academic medicine. Circulation **20:** 321, 1959.

Memoir of George Morris Piersol 1880–1966*

By T. GRIER MILLER, M.D.

GEORGE MORRIS PIERSOL, a former treasurer of our College and one of Philadelphia's outstanding physicians, died on August 19, 1966. He was born on October 13, 1880 and became the representative of a third generation of physicians in his family. His father was the distinguished Dr. George A. Piersol, who succeeded Dr. Joseph Leidy as Professor of Anatomy at the University of Pennsylvania. I, as a first year medical student, well remember him as my most fascinating teacher. Both of the father's parents were physicians, Morris' grandmother being one of the first graduates of the Woman's Medical College of Pennsylvania. Such a background surely was an important factor in the notable accomplishments of Morris Piersol as a clinician, teacher, scholar, editor, historian and administrator.

Morris grew up in Philadelphia, graduated from the William Penn Charter School in 1898 and subsequently received from the University of Pennsylvania a B.S. degree in 1902 and an M.D. degree in 1905. Following some postgraduate studies at the University of Groningen, Netherlands, he interned at the Hospital of the University of Pennsylvania and was its Chief Resident in 1906–7. Later, in the University's School of Medicine, he became an Instructor, an Associate, a Professor of Clinical Medicine (1942–47) and then, in 1947, its first Professor of Physical Medicine and Rehabilitation. At the same time he was made the Director of a Center in the hospital for instruction and research in the latter field. Meantime, in 1920, he had become Professor of Medicine in the University's Graduate School of Medicine, and eventually, from 1954 to 1957, served as its Dean. From 1920 to 1945 he was Medical Director of the Bell Telephone Company of Pennsylvania. Also, early in his professional career he had taken up the private practice of medicine in the office of Dr. John H. Musser, one of the University's eminent Professors of Medicine, this leading to the acquisition of an extensive and profitable practice among the elite of the city. He did not allow this, however, to interfere with his other interests, and such activity soon was abandoned. In addition to his University commitments he was a Professor of Medicine in the Woman's Medical College (1917–23) and a physician to the Philadelphia General Hospital (1917–31), to the Methodist Hospital (1921–31), to the Presbyterian Hospital (1929–33) and to the Abington Memorial Hospital (1931–34). Later he became a Consultant to the Lankenau Hospital and, in 1945, a Consultant in Physical Medicine and Rehabilitation for the U. S. Veterans Administration.

Dr. Piersol developed an especial interest in the American College of Physicians, was its Secretary-General for 20 years, its President in 1933–34, served as the Chairman of its Credentials Committee for many years and finally, as its Historian, wrote the fascinating story of its founding, developments and accomplishments, published (1962) as the *Gateway of Honor*. He was a president of the Philadelphia County Medical Society (1922), the American Gastroenterological Association (1929), the

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American Clinical and Climatological Association (1931), the Medical Club of Philadelphia (1933), the Pennsylvania Academy of Physical Medicine (1948–49) and the American Institute of Medical Climatology (1959). He was a member of the Association of American Physicians, the Industrial Medical Association, the American Academy of Physical Medicine and the American Congress of Physical Medicine. Also he was a member of the Pathological Society of Philadelphia and the Philadelphia Pediatric Society.

During the First World War, Dr. Piersol was commissioned a Lieutenant Colonel in the Army. He served as Commanding Officer of Base Hospital 20, the University of Pennsylvania unit, and later as Medical Consultant to the IV Army Corps of the A.E.F. in Europe. Among the many civilian honors bestowed upon him were a Mastership (1948) and the Alfred Stengel Award (1951) by the American College of Physicians, the Gold Key Award of the American Congress of Physical Medicine and Rehabilitation (1954) and, by the University of Pennsylvania, its Alumni Award of Merit (1951) and an Honorary Doctor of Science degree (1958). Also, in 1960, the Rehabilitation Center of the University Hospital was named in his honor. Among his Philadelphia social clubs were the Union League, the Rittenhouse, the Racquet, the Rose Tree Fox Hunting and the Corinthian Yacht. Of the latter he was the Fleet Surgeon for 29 years. He was a member of four Greek Letter fraternities.

Although Dr. Piersol's training was primarily along clinical lines and antedated the current enthusiasm and facilities for research, he kept abreast of all achievements in the field of medicine and made numerous significant contributions to leading medical journals. He recognized the impact that scientific developments were having on the fundamental aspects of clinical problems. He was especially influential in bringing new knowledge to the attention of the profession. This is manifest in his editorial accomplishments. For 11 years (1911–22) he was the Editor of the *American Journal of the Medical Sciences* and for 10 years (1937–47) of *International Clinics.* In addition he was the Editor-in-Chief of the *Cyclopedia of Medicine, Surgery and Specialties* (1939–64) and was on the Editorial Board of the *Archives of Physical Medicine and Rehabilitation* (6 years) and of the *Journal of Physical Medicine.*

Dr. Piersol also recognized the necessity of organizational approaches to the solutions of medical problems. He played an important part in the emergence of internal medicine as a specialty. This was demonstrated by his influence with colleagues in the problems of the American College of Physicians. His unique personal qualifications were a large factor in this. He had a charming personality, was gentle, humorous and tolerant. He was persuasive without being controversial. Kindness was an outstanding characteristic. He had no enemies and all acquaintances were his friends. He was ready at all times to render service to others, even to the point of sacrifice. In spite of physical handicaps in his later years he maintained to the end a warm, kindly and generous nature, had no complaints and died peacefully.

George Morris Piersol leaves behind many devoted friends, a faithful and dedicated wife, whom he married as Helen Delano on September 26, 1908, and three children: Helen (Mrs. George F. Price), Anne (Mrs. Edward A. Collins), and George Morris, Jr.

Memoir of Adolph A. Walkling 1895–1966*

By GEORGE WILLAUER, M.D.

REMINDED of Adolph A. Walkling, those of us who knew him well think of him as a Friend, a member of the Society of Friends. At the same time, one cannot refrain from associating this man with the thought of friendliness. It is about these two words that I want to talk with you for a few minutes.

Friends believe in God's illumination of every individual. In other words, everyone possesses what they call the Inner Light. I think that Adolph Walkling's embodiment of the Inner Light and his friendliness are synonymous. Let me give you two examples: Many years ago, in June of 1923, a young physician was working with Walkling in the surgical out-patient department of Jefferson Hospital. The apprentice admired the bandage scissors that Adolph Walkling was using. With alacrity and a friendly gesture, Walkling gave the scissors to the doctor who carries them with him this very day. More than forty years later, when Adolph Walkling lay in the intensive care unit of the hospital receiving intermittent positive pressure and intravenous therapy, he greeted his most recent resident with an inquiry about his wife and new-born baby. With further disregard of his own distress, Walkling reminded this doctor, who was, of course, in the early part of his surgical career, that the fruits of his labors would flourish. Adolph Walkling was gone within twenty-four hours. These to me are fine illustrations of charity and concern for others.

The two qualities of Friend and friendliness encompassed Walkling's whole life. His joys and adversities were shared by his devoted wife, the former Marian Ware. To them was granted the blessing of two fine sons, and theirs was a happy family relationship.

Like all proud fathers, Walkling had an intense interest in his sons. He was enthusiastic about their Scout maneuvers and school sports. He was also very mindful of their education. However, neither one chose the field of medicine. Richard became interested in electronics and is now manufacturing highly specialized electronic devices and components. Robert chose the academic life and is presently a professor of physics at Bowdoin College.

Walkling's hobbies were many and his enthusiasm for collecting antiques, rare books and prints, especially of the Pennsylvania Hospital, was shared by his family. With the rose petals which they gathered from their garden, the Walklings made potpourri for their unique and large collection of rose jars.

All of Walkling's professional life was spent in the teaching of students, interns and residents on the surgical services at the Jefferson and Pennsylvania hospitals. This friend to all was scrupulous in the technical care of his patients, manifesting the humanitarianism of the true physician. His skill and experience as a surgeon was recognized by his colleagues. He was further honored by being appointed as consultant in our various city and suburban hospitals.

Walkling was a member of many societies and was particularly interested in the affairs of the Philadelphia Academy of

* Read before The College of Physicians of Philadelphia, 7 December 1966. Prepared and published at the request of the Council of The College of Physicians of Philadelphia.

Surgery, serving as president in 1958–60, the American Surgical Association, the American College of Surgeons and our own College of Physicians. He contributed thirty-three papers to the surgical literature.

You may be aware that the concept of friendliness as we know it is not emphasized in the Bible. Here commitment and sacrifice are stressed as qualities of a friend. Quoting from the familiar passage where Paul defines charity in Corinthians I, Chapter 13, verses 4–8:

"Charity suffereth long, and is kind; charity envieth not; charity vaunteth not itself, is not puffed up,

Doth not behave itself unseemly, seeketh not her own, is not easily provoked, thinketh no evil;

Rejoiceth not in iniquity, but rejoiceth in the truth:

Beareth all things, believeth all things, hopeth all things, endureth all things.

Charity never faileth."

These truths approximate the life of our friend, Adolph Walkling.

Memoir of Max Bernard Lurie 1893–1966*

By ESMOND R. LONG, M.D.

MAX BERNARD LURIE, experimental pathologist, noted for his investigations of hereditary and constitutional factors in tuberculosis and of the mechanisms of resistance to infectious disease, died in Philadelphia on September 23, 1966. He had been in failing health for many months with increasing disability of the heart, but he had maintained his productivity to the end. With remarkable industry, he had devoted his final years to an extended review of pertinent work in his chosen field of resistance to tuberculosis, integrating his own long years of experimentation and experience in the accumulation of research on that subject.

Lurie was born on September 12, 1893, the second in a large family of children, in Telshee, Lithuania, then a part of the Russian empire. His family emigrated to the United States in 1907 in search of a freedom they were unable to enjoy in their native land. Lurie's parents were of distinguished Jewish ancestry, tracing their origins as far back as eleventh century Talmudic scholars. His father was a rabbi, and for a time young Lurie was expected to follow in his father's profession in America. Lurie himself was attracted rather to the field of philosophy. Medicine, likewise, had a strong appeal to him as a field in which he could be effective. He entered the Medical School of Cornell University, working his way through, and was graduated in 1921.

During his medical school days it became apparent that he had contracted pulmonary tuberculosis, possibly from family contagion. His mother died of the disease. After graduation he entered the National Jewish Hospital for Consumptives in Denver, Colorado, for care, and, as it turned out, for training in tuberculosis research. His innate resistance to the disease was apparently high; he overcame the infection and soon became a laboratory investigator of great promise on the hospital staff.

In 1926 he accepted an invitation from the Henry Phipps Institute of the University of Pennsylvania in Philadelphia to become a Fellow in Pathology under the Institute's director of research, Eugene L. Opie. He made a rapid start on laboratory investigation in the field of immunology, in which Opie and other members of the staff were deeply interested. The atmosphere was congenial, and Lurie had an independence in the direction of his efforts that he strongly desired. He remained at the Phipps Institute for forty years, attaining the rank of full professor in 1955. He worked with consummate industry and unfailing attention to detail, storing away the results of experience that made him a leader in his field in later years, with unusual perspective on knowledge accumulated during his time.

Germane to his major problem, the mechanism of resistance to tuberculosis, was exact determination of the mechanisms of infection itself, a field with which Lurie's chief, Dr. Opie, was much concerned as a clinical problem. Lurie, with ingenuity, and imagination as well, set up experimental models in which the transmission of infection from infected to healthy guinea-pigs in cages could be

* Prepared and published at the request of the Council of The College of Physicians of Philadelphia.

followed with precision. These experiments and subsequent more elaborate ones, in which ultraviolet light was used to sterilize air, in which he was associated with the hygienist, W. F. Wells, have had wide application in places of public gathering.

In 1932 Lurie acquired a colony of highly inbred rabbits from a stock raised for experiments at the University of Chicago, from which he developed a strong foundation for his later studies. By litter mating he soon separated out substrains of animals with differing degrees of resistance to tuberculosis. The extremes among these substrains corresponded closely, in the type of tuberculosis developing in them, with variations in susceptibility and resistance as seen in spontaneous tuberculosis in man. Continuing with a steady program of inbreeding in the colony, he was soon able to determine specific physiological and cellular differences in these animals, accounting for their individual degrees of resistance. He devoted a great deal of attention to the role of the macrophages in the different animals. His studies in that field are recognized today as fundamental in widely diversified and productive studies, in many lands, now under way.

Studies of the mechanisms involved led him in time to investigations of the controlling influences of the body's internal secretions on the response to infection. In his latest investigations he uncovered striking differences in the effects of two endocrine systems, the cortico-adrenal hormones and thyroid hormone derivatives. The significant elements of this long research were embodied in Lurie's now well known monograph, "Resistance to Tuberculosis: Experimental Studies in Native and Acquired Defensive Mechanisms". This was brought out in 1964 under the auspices of the Commonwealth Fund, which had supported much of his work over the years.

Many honors were conferred on Lurie, including the Dearbolt lectureship in Wisconsin in 1952, the Bernard Medal of the University of Montreal in 1953, and the Trudeau Medal of the National Tuberculosis Association in 1956. He was elected a Fellow of The College of Physicians of Philadelphia in 1947. He was a regular attendant at scientific meetings of the College, and well informed on the presentations made there. He was active in the scientific sessions of the National Tuberculosis Association and faithful in completing assignments by that Association in research and editorial fields. He was awarded a life membership by the New York Academy of Sciences.

Lurie's major impact on medical research was in the exposition of inherent, hereditary factors as influential and determinative in resistance to tuberculosis. He was highly successful in correlating these factors with the competence of the body's macrophages in resistance and susceptibility. He was an excellent teacher, popular for his enthusiasm, among students of the University of Pennsylvania during their instruction at the Phipps Institute. He was noted for the intensity of feeling he displayed in his presentation of research in medical meetings.

Lurie is survived by his wife, Rose Gamoran Lurie, an educator and author in Jewish history, and a son, A. A. Lurie, now Associate Professor of Anesthesiology at the University of Rochester. Their home was a happy one, visited by many friends. Lurie was a serious conversationalist, never trivial. He had a strong feeling of *Weltschmerz,* but was basically optimistic. He was an idealist always, in research or out of it.

Memoir of Stephen Dana Weeder 1896–1966*

By JAMES S. C. HARRIS, M.D.

STEPHEN DANA WEEDER was born February 18, 1896 in Philadelphia, Pennsylvania. He attended Central High School and the University of Pennsylvania School of Medicine, from which he was graduated in 1917. His internship was deferred while he served in the United States Army from 1917–19, spending 19 months in Europe and attaining the rank of Captain in the Medical Corps. After his discharge from the Army he served a rotating internship at Philadelphia General Hospital for 18 months.

Dr. Weeder then started on a distinguished career in surgery. He was appointed to the staffs of the Germantown and Chestnut Hill Hospitals and spent several years at Pennsylvania Hospital. He spoke on many occasions of his preceptors, Drs. William B. Swartley and Robert Le Conte, and their influence upon him. His teaching years began in 1920 at the Department of Anatomy of the University of Pennsylvania School of Medicine where he held a post until 1932. From 1942–46 he was a Clinical Associate Professor of Surgery at Woman's Medical College, and continued as Clinical Professor until 1949. At that time he was appointed a Clinical Professor of Surgery at Jefferson Medical College and continued his teaching of the Jefferson medical students until 1961.

Dr. Weeder was certified by the American Board of Surgery in 1938. He was appointed Director of the Department of Surgery of both Germantown and Chestnut Hill Hospitals, and later served as president of the staffs of these two institutions for several terms, twice at Germantown and four times at Chestnut Hill Hospital.

Of the many organizations of which he was a member, he felt that Philadelphia Academy of Surgery was the finest. He was a member of the Founders' Group of this Academy in 1928 and served as treasurer from 1947–60. He was a Fellow of The College of Physicians of Philadelphia, and he served as a member of the Board of Directors of the Philadelphia County Medical Society from 1946–50. He was a member of the American College of Surgeons and the American Medical Association.

Research was another important facet of his life. Dr. Weeder spent considerable effort in the experimental production of regional enteritis and in studying its relationship to mesenteric lymphadenitis. Later he showed his diversity by developing a direction finder for fractures of the femoral neck.

His writings and publications were many, covering a wide area. He published two editions of Bundy's *Anatomy and Physiology*. He also was an assistant editor of the 9th Edition of Davis' *Applied Anatomy* revised by Dr. George P. Muller.

Dr. Weeder married Caroline D. Nixon on April 16, 1925 and they had four children, two girls, Dorothy and Caroline, and two sons, Dana and Richard. Both of his sons are now in surgery. Dana finished his residency at the Germantown Hospital and is now serving with the United States Navy as a Lieutenant Commander in Vietnam. Richard is completing his final year of surgery residency at the Geisinger Memorial Hospital in Danville, Pennsylvania.

* Prepared and published at the request of the Council of The College of Physicians of Philadelphia.

Dr. Weeder was a great sportsman—squash in his young years and golf forever. He was a strong member of the Philadelphia Doctors Golf Association and often said that he would rather play a sport than observe it. Sailing, especially yacht racing, was another favorite sport, and he never attained his fill of it.

One cannot conclude a Memorial without recalling Dr. Weeder's great dedication to surgery, the hours he gave to teaching, to the patients and to his residents. Time was never a factor in his desire to demonstrate and pass on to the younger men the basic principles of surgery. Likewise basic principles in *all* of life were very fundamental to him and his emphasis in this direction, with his associates and children, left a mark that will never be forgotten.

He was a devout Presbyterian and prayer was always a segment of his life.

Dr. Weeder died quietly at home on October 5, 1966, leaving a strong family and many friends stronger by his integrity and fundamental ideals.

Memoir of William Oscar LaMotte, Sr. 1880–1966*

By FRANCIS HEED ADLER, M.D.

WILLIAM O. LAMOTTE, SR., died in Wilmington, Delaware, on October 5, 1966 at the age of 86 years, having practiced medicine for over fifty years. He had a varied and colorful life, entailing many diverse interests, such as running a farm (the one on which he was born in Western Maryland), boxing, teaching school, writing letters to the editors of newspapers giving his ideas on political, legislative and civic issues, and practicing ophthalmology.

He had early leanings toward the military services and attended St. John's College in Annapolis. He sent in his application papers for a commission, but the papers were lost. A second lot of papers was laboriously prepared, but was destroyed in a fire. He then decided with good humor that the Fates must be directing him into other channels, and he became a school teacher. Eventually he became principal of a high school in Hampstead, Maryland. After acquiring sufficient funds, he decided to study medicine and entered Johns Hopkins University. However, he was unhappy there, and transferred to the School of Medicine of the University of Pennsylvania. During this period he became interested in the manly art of self-defense, as it used to be known, and acquired sufficient expertise to work out in the city's gymnasiums with professional prize fighters.

After his graduation in 1907 he interned at St. Agnes' Hospital and opened an office for general practice in Riverside, New Jersey. For five or six years he also commuted back and forth to Philadelphia, working with eye, ear, nose and throat specialists, taking post-graduate courses at the old Polyclinic Hospital and attending clinics abroad. Eventually he gave up general practice and confined his work to EENT. About fifteen years before he died, he limited his practice to ophthalmology, and continued to see patients until his death.

One of his chief joys in life was to see his son, William O. LaMotte, Jr., enjoy the opportunities and succeed in the one field of medicine which really interested him, ophthalmology. It was in this connection I became personally acquainted with Dr. LaMotte, Sr., for his son took his residency with me at the Hospital of the University of Pennsylvania. During these years, unknown to his son, Dr. LaMotte, Sr., started taking the Home Study Courses of the American Academy of Ophthalmology and Otolaryngology, travelling here and there around the country to take courses generally given for younger men, and eventually equipping himself so that at the age of 63 years he presented himself for the examination of the American Board of Ophthalmology and received its diploma—a very remarkable achievement for a man so many years out of medical school and without any formal residency training. This was one of the great satisfactions of his life; the other was his pride in seeing his son become an outstanding ophthalmic surgeon.

In 1914 Dr. LaMotte, Sr., came to Wilmington, and at one time was President of the Staff of the Delaware Hospital (now Delaware Division of the Wilmington Medical Center). He was also Chief of the

* Prepared and published at the request of the Council of The College of Physicians of Philadelphia.

Ophthalmological Clinic and Assistant Ophthalmic Surgeon at the Presbyterian Hospital in Philadelphia. He was very active in Medical Society affairs at the county, state and national level. He served as President and Secretary of the Medical Society of Delaware, was a member of the House of Delegates of the American Medical Association and was a charter member of the Delaware Academy of Medicine. He was a member of the American College of Surgeons, The College of Physicians of Philadelphia, and the American Academy of Ophthalmology and Otolaryngology.

He is survived by his widow, Mrs. Sue Clary Hall LaMotte, whom he married in 1911, his son, Dr. William O. LaMotte, Jr., and a daughter, Mrs. Robert F. Deese.

Memoir of Samuel Timothy Nicholson, 2nd 1887–1966*

By T. GRIER MILLER, M.D.

SAMUEL TIMOTHY NICHOLSON, 2nd, of Pottstown, Pennsylvania, a Non-Resident Fellow of our College since 1937, died on May 8, 1966. Though born in the home of a grandmother in Norfolk, Virginia, he really was a native of Bath, North Carolina, where his father, Dr. John Thorne Nicholson and two of his father's brothers were practicing physicians. His mother was Katherine McNeal Nicholson, a Virginian. He had three brothers: John Thorne, Jr., Edward Harold and William M., the latter now a Professor of Medicine at Duke University. Also he had two sisters, Blanche Baker (Webb) and Mary Lucille (Wolf), both now widows. He married Mary Cater Peyton of Pulaski, Virginia, on November 4, 1911, the wedding taking place in New Castle, Delaware. She survives him. They had two children, Charles Peyton, who is district manager of Maxwell House Coffee, and Samuel T., 3rd, who, as a 2nd Lieutenant in the Marine Corps, was killed on Iwo Jima on March 7, 1945.

Until ten years of age, Sam, also called "Nick" by his friends, was tutored in his home and then attended the Graham Preparatory School in Warrenton, North Carolina. Subsequently he entered the University of North Carolina, where, with me, he was graduated in 1906. He obtained his M.D. degree from Johns Hopkins in 1910. He interned in the Telfair Hospital in Savannah, Georgia. Then he spent a year with Dr. Kenneth D. Blackfan in a Pediatric Laboratory at Washington University in St. Louis, Missouri. In 1912 he became associated with the Municipal Hospital in Baltimore, but, because of its political affiliations, he resigned and accepted, in March, 1914, an invitation to join the staff of the Clifton Springs Sanitarium in New York State. Except for his service in the Medical Corps of the Army at Fort Riley, Kansas, during the First World War, he remained there until 1923, when he became Medical Director of the Hill School at Pottstown. At about the same time another Hopkins graduate, Dr. C. S. Guthrie, became Medical Director of the Lawrenceville Academy in New Jersey. The two of them were the first physicians in this country to inaugurate preventive medicine in secondary schools, now routine in most such schools. The new challenge greatly appealed to Sam Nicholson and his success in this endeavor constituted his most important contribution to medicine. Upon his retirement from the Hill School in 1948 he remained in Pottstown and until 1957 was the Medical Director of the Pottstown Plant of the Doeher Jarvis Corporation, a producer of die castings.

Although never connected with any teaching or research medical institution Dr. Nicholson, especially in his younger days, kept in close touch with the advances in medical knowledge and indeed made some contributions. He was the author or co-author of 21 articles in medical journals. His first one, in 1913, with Dr. Blackfan was "A Study of the Wassermann Reaction in One Hundred Infants". Subsequently he published four other papers on syphilis, three on diabetes, with special reference to the use of insulin, and one each on men-

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ingitis secondary to sinusitis, angina in poliomyelitis, infectious arthritis and anemia following transfusions.

In addition to his membership in the American Medical Association and our College, he was a member of the American Clinical and Climatological Association (1927) and the American College of Physicians (1929). Also he received an Award from the Royal Society of Health of Great Britain.

During his tenure of office at the Hill School, I, on a consultant basis, saw many student patients with Dr. Nicholson. I always was impressed by his medical knowledge, his diagnostic ability and especially his deep interest in the human aspects of his problems. He was a well trained and highly efficient physician, understood human nature and got along nicely with his non-medical associates as well as his patients and their families. He had a pleasing personality, was kind and generous in his relations with family and friends and had a real sense of humor. A classmate of his at Hopkins, Dr. L. Whittington Gorham, has said of him, "He was the youngest member of his class and no one was more popular or more highly respected." He was an Episcopalian and deeply religious, but more interested in its social, moral and intellectual aspects than its emotional ones. His hobbies were reading, golf and gardening, especially the growing of roses.

A malignant pulmonary lesion was discovered in 1961, too advanced for radical therapy, but, with radiological and chemical treatment and in spite of marked incapacity during his later years, he maintained an optimistic and cheerful attitude until the date of his death.

Report of the Secretary for the Year Ended December 31, 1966

		Total Attendance
Stated Scientific Meetings:		
January	The Fiftieth Nathan Lewis Hatfield Lecture Seymour S. Cohen, PH.D.	56
February	The Seventy-eighth Thomas Dent Mütter Lecture Averill A. Liebow, M.D.	117
March	The Forty-fifth James M. Anders Lecture Luigi Mastroianni, Jr., M.D.	55
April	The Ninth Benjamin Musser Lecture on Clinical Medicine Theodore E. Woodward, M.D.	72
May	The First Theodore F. Bach Memorial Lecture Currier McEwen, M.D.	70
	At the Philadelphia County Medical Society. Presentation of the Second Annual Kenneth E. Appel, M.D., Award to Melvin Singer, M.D., Resident in Psychiatry, Institute of Pennsylvania Hospital.	
	Presentation of Certificates to Physicians who have completed fifty years in the practice of medicine by the President of the Pennsylvania Medical Society.	
	Presentation of the Strittmatter Award to Eugene P. Pendergrass, M.D., Emeritus Professor of Radiology, University of Pennsylvania School of Medicine.	
October	The Ninety-first Mary Scott Newbold Lecture Charles R. Scriver, M.D., F.R.C.P. (Can.)	62
November	Combined meeting with the Philadelphia County Medical Society and the Pathological Society of Philadelphia at the Hall of The College of Physicians.	
	The Seventy-ninth Thomas Dent Mütter Lecture Donald G. McKay, M.D.	201
December	The Twenty-sixth Alvarenga Lecture Hans Popper, M.D.	97

Regular Business Meetings:	*Total Ballot*
January 5, 1966	371
February 2, 1966	241
March 2, 1966	229
April 6, 1966	261
May 4, 1966	243
October 5, 1966	301

November 2, 1966	268
December 7, 1966	292
	Ballot for Amendment
May 4, 1966	235

The College of Physicians of Philadelphia awarded the Alvarenga Prize Award on July 14, 1966, to Hans Popper, M.D. (Pathology), for his outstanding work on Organelle Pathology of the Liver.

Sections of the College	*Membership*
Ophthalmology	80
Otolaryngology	42 & 4 Honorary & 4 Dues Remitted
Public Health, Preventive & Industrial Medicine	68
Medical History (Fellows & Friends of the Section)	120

Membership:

	Fellow	Non-Res.	Assoc.	Honorary Foreign	Honorary U.S.	Hon. Assoc.	Total
December 28, 1965	1087	158		2	6	3	1256
Died during year	25	3			2	1	31
Resigned	9	2					11
Elected	62	2					64
Transferred from Resident to Non-Resident	−9	+9					
Transferred from Non-Resident to Resident	+2	−2					
Failed to qualify	1						−1
Forfeited	2	1					3
December 31, 1966	1105	161	0	2	4	2	1274
Life Membership							
New Type							
Under 70	21	6					
Over 70	25	7					
Old Type (Commutation dues)	6	0					
	52	13					
Dues Remitted							
Before 1966	45	10					
During 1966	2	0					
	47	10					
Annual Contributors as of 12-31-66	1006	138					

GEORGE A. HAHN, M.D.
Secretary

Report of the Treasurer for the Year Ended June 30, 1966

Assets

General Fund:			
Cash in bank and on hand			$ 42,434.50
Maintenance and Reserve Fund:			
Cash in bank		$ 1,123.18	
Investments		16,409.88	
			17,533.06
Joseph N. Pew. Jr., Memorial Loan Fund:			
Investments		40,402.19	
Notes receivable		19,400.00	
			59,802.19
Employee Pension Plan:			
Investment with Insurance Company of North America			33,691.05
Trust Funds:			
Uninvested principal cash		27,133.63	
Uninvested income cash (restricted)		31,888.47	
Mortgage		28,125.00	
Real estate		158,905.33	
Bonds		590,695.14	
Preferred stocks		235,245.43	
Common stocks		857,589.19	
			1,929,582.19
Plant Fund:			
Land—16-25 South 22nd Street		95,000.00	
Building		1,420,747.93	
Furniture and fixtures		1.00	
Deposits on perpetual fire insurance policies		11,462.28	
			1,527,211.21
			$3,610,254.20

Funds

General Fund:			
Library general account		$ 16,420.95	
Library medical documentation service		14,100.94	
Library special account		17,356.01	
Life membership account		1,895.00	
Reserve for contingencies—Uninvested funds		190.02	
Greater Philadelphia Committee for Medical-Pharmaceutical Science		6,613.21	
Unexpended operating fund balances:			
Current operations—overexpended	($ 19,451.79)		
Committee on Interns and Residents	717.89		
President's room	525.00		
Books	1,649.09		
Rare books	100.00		
Compaign expense	85.95		
Portrait	126.47		
Friends of the Library	1,265.00		
Reception—Academy of Music	569.37		
Section on General Medicine	271.39	(14,141.63)	$ 42,434.50

Maintenance and Reserve Fund—principal			17,533.06
Joseph N. Pew, Jr., Memorial Loan Fund—principal			59,802.19
Employee Pension Plan—principal			33,691.05
Trust Funds:			
Principal net worth			
Discretionary common trust fund	1,671.737.19		
Legal common trust fund	24,097.94		
Trust funds not pooled	14,828.26		
Fund for purchase of units in discretionary common trust fund	28,125.00		
Real estate fund	158,905.33	1,897,693.72	
Income net worth			
Discretionary common trust fund	27,631.64		
Legal common trust fund	1,242.16		
Trust fund not pooled	3,014.67	31,888.47	1,929,582.19
Plant fund—principal net worth			1,527,211.21
			$3,610,254.20

NOTES: The various fund investments are shown at their recorded values, which values substantially represent cost.

GENERAL FUND

STATEMENT OF CASH RECEIPTS AND DISBURSEMENTS

Year Ended June 30, 1966

Receipts:			
Income on investments, net of agents fees:			
Trust funds	$86,816.90		
Less: Amount transferred to income of the several restricted trust funds	13,932.46	$72,884.44	
Erlanger Garage			
Income	10,621.99		
Expenses	5,661.47	4,960.52	
Income from funds, principal of which is not administered by College	2,699.00		
Less, amount transferred to income of a restricted fund	478.48	2,220.52	
Interest on mortgage	1,328.87		
Income from other sources	1,158.08	2,486.95	$82,552. 4
Contributions:			
Fellows dues		87,233.96	
Life membership		1,965.00	
Use of hall and projector		3,198.00	
Friends of Library		1,265.00	
Reception—Art Museum		3,340.00	
Section on General Medicine		271.39	
For Greater Philadelphia Committee for Medical-Pharmaceutical Science		9,500.00	106,773.35
Transfers from other funds:			
For reducing deficit in current operating account:			
Miscellaneous Contributions Fund		2,749.35	
For covering deficit of Entertainment Committee:			
S. Weir Mitchell Entertainment Fund		398.06	3,147.41
Library:			
Contribution plan	24.365.50		
Photostats, fines, etc.	40,978.82		
Transfer from library special account	5,896.49	71,240.81	

Medical documentation service		47,621.74	
Special account—influenza grant		15,318.00	134,180.55
Miscellaneous			2,417.25
Total receipts			329,070.99
Disbursements:			
Operating expenses:			
Hall Committee		$ 38,820.19	
Office of the Secretary		8,479.17	
Office of the Treasurer		12,455.09	
Old Age Benefit taxes		5,499.88	
Pensions		488.00	
Compaign expenses		10.00	
Publication Committee		9,662.69	
Books		403.39	
Entertainment Committee		2.019.56	
College Collections Committee		4,276.46	
Premium to Pension Fund		6,497.00	
Reception—Art Museum		2,770.63	
Library General:			
Salaries	$111,909.73		
Books, journals, supplies, etc	54,380.78	166,290.51	
Medical documentation:			
Salaries	22,311.87		
Other expenses	11,208.93	33.520.80	
Library Special:			
Journal acquisition fund	4,851.18		
Influenza grant	6,998.00	11,849.18	
Greater Philadelphia Committee for Medical-Pharmaceutical Science		12,648.72	
		315,691.27	
Erlanger Garage profit applied to deficit of July 1, 1965		861.19	
			$316,552.46
Excess of receipts over disbursements			12,518.53
Cash balance, June 30, 1965			29,915.97
Cash balance, June 30, 1966			$ 42,434.50

Statement of Cost of Investment Units Purchased in the Various Trust Funds by the Restricted Endowment Funds

July 2, 1965 to July 1, 1966

Discretionary Common Trust Fund:		
Balance, July 2, 1965		$722,373.52
Receipts:		
S. Weir Mitchell Associates journal fund	$12,197.74	
Edward B. Krumbhaar Book Fund	1,000.00	
Life Membership Fund	1,044.97	
Transfers from unit adjustment accounts:		
S. Weir Mitchell Associates journal fund	21.33	
Life Membership Fund	21.33	
	14,285.37	
Less: Amounts insufficient to purchase one unit, credited to unit adjustment account	36.93	
		14,248.44
Balance, July 1, 1966		736,621.96
Legal Common Trust Fund:		
Balance, July 1, 1965 (no change during year)		23,500.00

Trust Funds Not Pooled:	
Balance, July 1, 1965 (no change during year)	14,974.56
Total restricted endowment funds, July 1, 1966	$775,096.52

STATEMENT OF COST OF INVESTMENT UNITS PURCHASED IN THE DISCRETIONARY COMMON TRUST FUND BY THE GENERAL ENDOWMENT FUND

July 2, 1965 to July 1, 1966

Balance, July 2, 1965		$ 493,481.77
Less: retirement of units upon removing Erlanger Garage from Discretionary Common Trust Fund, November 30, 1965		158,906.84
		334,574.93
Receipts:		
Entrance contributions	$ 2,900.00	
Voluntary contributions from Fellows, etc.	801.85	
Book of Remembrance	170.00	
Amortization of mortgage—13th and Locust Streets	2,250.00	
Estate of Damon B. Pfeiffer	50,000.00	
Estate of William Zentmayer	425.88	
	56,547.73	
Less: Amount insufficient to purchase one unit, credited to unit adjustment account	1.90	56,545.83
Balance, July 1, 1966		$ 391,120.76

STATEMENT OF COST OF INVESTMENT UNITS PURCHASED IN THE DISCRETIONARY COMMON TRUST FUND BY THE COLLEGE EXTENSION FUND

July 2, 1965 to July 1, 1966

Balance, July 2, 1965		$ 4,334.63
Receipts:		
Voluntary contributions from Fellows	$ 215.00	
Income earned on invested principal	229.18	
Transfer from unit adjustment account	21.33	
	465.51	
Less: Amount insufficient to purchase one unit, credited to unit adjustment account	17.58	
		447.93
Balance, July 1, 1966		$ 4,782.56
Total of all restricted funds, July 1, 1966		$1,170,999.84

KENDALL A. ELSOM, M.D.
Treasurer

Annual Report on the Library*

The composition of an annual report in February 1967 for publication in the April 1967 issue of *Transactions & Studies of The College of Physicians of Philadelphia,* covering a report year running from sixteen to four months previous, opens its authors to a number of temptations. Enthusiasm for current events and plans for the future compete for the attention required to chronicle the past properly. It is natural to write implications into the record that are realized only in the light of developments which occurred after the year closed. It is also illogical to preserve a report year originally designed to coincide with a fiscal year which has now been changed to a different span. Not only does the present report year differ from the fiscal year but it does not coincide with the calendar year terms of the officers and committees of the College. Our resolve to limit the following report to the proper twelve month period may have been slightly bent in spots.

The Perspective. The excitement generated by the passage of the Medical Library Assistance Act of 1965 stimulated a broad perspective of the potential role of the College Library in a nationwide system of medical libraries. This year's report concerns itself with major activities which reflect this broad perspective.

Proposed Cooperative Agreement. The application form for Medical Library Resource Grants provided space for indication of "cooperative agreements relating to the sharing of resources". This reference, together with the knowledge that, if funds permitted, such sharing agreements might lead to augmentation of the Resource Grant itself, led the College Library to seek a modest formalization of existing informal relationships.

The Proposed Cooperative Agreement between The College of Physicians of Philadelphia Library and Philadelphia Area Medical Libraries was first framed during the early spring of 1966 and it was then strengthened after consultation with the Regional Medical Library Committee. It was distributed during the summer and had been endorsed by forty-five local medical institutions at the time of this writing.

Following a brief historical statement of its background, the agreement presents the College Library's recognition of its responsibility to share its resources with other medical libraries to the fullest extent possible, consistent with maintaining high standards of service to the entire community. The College Library's service and acquisition policy has been predicated upon its responsibility to serve as a regional library. Its policies have been influenced by the subject strengths and weaknesses of neighboring institutions. It is more important to augment existing resources than to duplicate them. Circulation forms, policies, and routines are designed to expedite service to the entire community.

Cooperating libraries, endorsing the Cooperative Agreement, recognize their dual responsibility to the entire community as well as to their own clientele. They acknowledge a moral obligation to share the costs of the College Library's service to them to the best of their ability without jeopardizing the ability to serve their own users. Cooperating libraries with well-developed subject strengths in specialized areas recognize a responsibility to develop greater depth than is required by their own clientele to augment the total strength of medical libraries in the area. They also indicate their willingness to share resources with each other as freely as is consistent with high standards of service to their immediate constituency.

As far as we know, no other area of the

* November 1, 1965—October 31, 1966.

country has provided a similar testimony to its determination to cooperate fully to provide the best possible medical library service to an entire community.

Medical Library Resource Grant Application. Further demonstration of the community-minded attitude of the College Library was afforded by its application for a Resource Grant under the Medical Library Assistance Act. Emphasis in this application is placed on increasing and improving service to the medical professions. The College requested funds to provide free photocopy in lieu of interlibrary loan to medical school and hospital libraries. It is hoped thereby to speed delivery of information since daily mail will be quicker than occasional messenger service and to increase the accessibility of articles since original volumes remaining in the library can be copied again or can be consulted which they can not be when they are in circulation.

The Resource Grant proposal also requests support for a teletype connection with the National Library of Medicine which will enable the College Library to secure material not available in the Philadelphia area. Requests made on behalf of other libraries may be sent directly from Bethesda to the requesting library without passing through the College Library.

Any funds remaining in the grant will be devoted to strengthening the Library's book and journal acquisitions. It must be emphasized that this particular grant does not in any way ameliorate the College's own financial problems. The grant is limited to support of services not now offered by the Library and it does not provide any allowance for overhead. Because of this it may even intensify the existing fiscal difficulty. Even more significant is the fact that the grant is self-reducing. It is made for a five-year period and is reduced by twenty percent each year. This means that the cost of the services provided must gradually be absorbed either by the College or by the recipients of the service.

MEDLARS site visit. The College Library gave further evidence of its desire to serve and of its faith in an automated future by informing the National Library of Medicine that it is willing to serve as a MEDLARS search center. A precisely detailed report was prepared to indicate the Library's ability to meet the qualifications for such a center as specified by the Board of Regents of the National Library of Medicine. This approach culminated on May 24, 1966 with a visit to the College by a team from the National Library of Medicine composed of Dr. Russell A. Dixon, D.D.S, Board of Regents; Dr. Joseph Leiter, PH.D., Associate Director for Intramural Programs; Paul C. Redmer, Acting Chief, Information Systems Division; Mrs. Yvonne Scott, MEDLARS Search Liaison Officer.

In the search for MEDLARS compatible equipment in the Philadelphia area, a Honeywell 800 installation was discovered at the Philadelphia Electric Company about two blocks from the College's front door. Without committing themselves to actual involvement, representatives from the Computer Division of the Philadelphia Electric Company demonstrated their sympathetic interest by attending a luncheon meeting on the day of the site visit and by exploring the requirements of the system in connection with the availability of their own resources. Other persons who met with the visiting team were: John F. Harvey, PH.D., Dean, School of Library Science, Drexel Institute of Technology; Richard H. Orr, M.D., Director, Institute for the Advancement of Medical Communication; John F. Huber, M.D., Chairman, Philadelphia Regional Medical Library Committee; The Honeywell Company was represented by Len Mayo of the Electronic Data Process Division. The case for the College was

presented by John H. Gibbon, Jr., M.D., President; S. X Radbill, M.D., Chairman, Library Committee and the Librarian, the Executive Secretary of the College and the Library's Department Heads.

Facsimile transmission grant application. Considerable effort was invested by personnel from the five Philadelphia medical schools and The College of Physicians in the preparation of a grant application to establish a facsimile transmission network between the College and these institutions. Representatives from the Xerox Corporation and the Bell Telephone Company were especially helpful in technical details. The librarians' enthusiasm was shared by the administrative officers of the six institutions. The endorsement of the governing bodies of all cooperating agencies was secured. Competent experience was drawn upon in the formulation of statistical and research aspects of the project which was intended to serve as a pilot study for similar installations in other parts of the country. On November 18th, shortly after the close of this report year, we learned that the Board of Regents Committee for Extramural Programs of the National Library of Medicine did not recommend approval of the application for a research grant.

Not surprisingly, the co-authors of the proposal remain convinced of its validity but have no plans for revising or resubmitting the proposal at the present time. Considerable comfort and inspiration were derived from the high level support which the planning librarians received and all those involved felt that they had learned a lot about design of experiments and the mechanics of grant applications.

Philadelphia Regional Medical Library Committee. This region's pioneering in medical library cooperation was demonstrated in 1963 when the Committee of the Deans of the Philadelphia Medical Schools asked the President of The College of Physicians of Philadelphia to appoint an interlibrary medical organization to seek means of establishing certain central facilities which could be shared by the five medical schools of Philadelphia and also other medical organizations. During the twelve-month period covered by this report, there were several meetings of this Committee of sufficient general interest to recount here.

In April 1966, the Committee was impressed by plans for the new library at Jefferson Medical College which were presented by Robert T. Lentz, Librarian. At this meeting the Committee endorsed the letter which The College of Physicians sent to the National Library of Medicine indicating its willingness to serve as a MEDLARS search center.

At this April meeting, members of the Regional Committee also received copies of the "letter of general intent" which was to be submitted by the Library of The College of Physicians of Philadelphia in response to the FACT SHEET on the MEDICAL LIBRARY RESOURCE GRANT PROGRAM issued by the National Library of Medicine. Much of the material in this "letter of general intent" was drawn from suggestions received at the Regional Medical Library Committee meeting held on April 27, 1965.

The Committee also reviewed the FACT SHEET. THE REGIONAL MEDICAL LIBRARY GRANT PROGRAM. Special attention was given to Section 13, What should libraries and institutions do now in preparation for the advent of the Regional Medical Library Grant Program?

In June 1966, the Regional Committee met to consider the Proposed Cooperative Agreement between The College of Physicians of Philadelphia Library and Philadelphia area Medical Libraries. As a result of this consideration, the Proposed Cooperative Agreement was strengthened and was endorsed by the Regional Committee.

At a meeting held on October 24, 1966, the Regional Committee reviewed the proposal of a Feasibility Study for a Health Sciences-Medical Library System to serve the Greater Delaware Valley Region prepared for the information of the University City Science Center Board of Directors Subcommittee on Health and Life Sciences. The Regional Medical Library Committee also discussed plans for a Regional Medical Library Conference to be held in January 1967 to fulfill the suggestions made in the Regional Medical Library Grant Program Fact Sheet which had been reviewed at the April meeting.

At this same meeting the Regional Committee authorized the Librarian of The College of Physicians to investigate the costs of a teletypewriter exchange service for interlibrary loan requests between the College Library and the National Library of Medicine.

Plans for the Regional Medical Library Conference to be held in January were developed in greater detail at a meeting held in November after the close of the current report year.

Members of the Regional Medical Library Committee during the year were: Miss Jacqueline Bastille, Librarian, Smith Kline & French Laboratories; Mrs. Lucy F. Cooke, Librarian, Hahnemann Medical College; Mrs. Ruth Diamond, Librarian, Temple University School of Medicine; Miss Ida Draeger, Librarian, Woman's Medical College; John F. Huber, M.D., (Chairman), Temple University School of Medicine; Marion Fay, PH.D., former Dean and President of Woman's Medical College; Miss Frances Houston and Mr. Jerome Rauch, University of Pennsylvania School of Medicine; Mrs. Helen Lake, Librarian, Philadelphia General Hospital; Mr. Robert T. Lentz, Librarian, Jefferson Medical College; Mr. Elliott H. Morse and Samuel X Radbill, M.D., College of Physicians of Philadelphia. After the close of the current report year, Mr. Richard L. Snyder, Director of Libraries, Drexel Institute of Technology and Mr. Fred Bryant, Librarian, Hershey Medical College accepted appointment to the Committee.

Library stack requirements. At the beginning of this report and again at its end, the Library Committee was considering the imminent need for expansion of the Library's stack space. Costs could vary from $180,000 to $400,000 depending on whether the building is extended to Van Pelt Street or whether two more floors are added to the present new stack unit.

Training medical librarians. The Chairman of the Library Committee and the Librarian have conferred with John F. Harvey, PH.D., Dean of the School of Library Science of Drexel Institute of Technology, concerning ways in which The College of Physicians Library might cooperate with Drexel in an expanded program of education for medical librarians. The National Library of Medicine has suggested that Drexel might help in training personnel and in the conduct of research bearing on the problem of biomedical information.

Friends of the Library. The establishment of the Friends of the College Library was announced in the last year's annual report. The group was launched at a Champagne Reception which was held at the Philadelphia Museum of Art in November 1965. No further activity was planned during the current report year.

Herner and Company Study. The Chairman of the Library Committee and the Librarian collaborated on an analysis of a "Recommended Design for the U. S. Medical Library and Information System" prepared under the auspices of the U. S. Office of Science and Technology. This study was intended to examine important questions such as the function, relationships to other libraries, operating procedures, resource requirements, etc., of a regional medical library in a total na-

tional network. Funding of the regional medical library section of the Medical Library Assistance Act of 1965 was delayed pending the preparation and analysis of this report.

Ten or more. The number of institutions which borrowed "ten or more" volumes from the College Library rose this year to 127 from a previous high of 110 last year. Ninety-one of these institutions increased their use of the College Library. The total number of volumes borrowed by this "ten or more" group rose this year from 21,555 volumes to 26,435 volumes, an increase of almost 25 percent.

Financial support. In the course of reviewing the long-range direction and financial well-being of the College, the Planning Committee has become interested in whether institutional users of the Library are adequately and equitably sharing the costs of operation. In July, Donald M. Pillsbury, M.D., Chairman of the Committee and John H. Gibbon, Jr., M.D., President, visited the New York Academy of Medicine to review its organization and various functions for purposes of comparison with the College. Many problems were found to be similar although the Academy has ten times the endowment and three times the membership of the College.

As the report year ended, The Greater Philadelphia Committee for Medical and Pharmaceutical Sciences indicated an interest in the Library's financial well being and a cost study of the Library was being contemplated by the Domestic Audits Department of Smith Kline & French Laboratories.

College Library publicity. An article in the September 5, 1966 issue of *Philadelphia Medicine,* written by George E. Farrar, Jr., M.D., under the title "Delaware Valley Right or Wrong" suggested that the College Library may become a mere monument to the past unless it is expanded "into the center for the storage and rapid retrieval and correlation of the bioscientific information required for the effective functioning of medical care, medical education and medical research here both now and in the future." The Chairman of the Library Committee and the Librarian, writing in the October 5th issue of *Philadelphia medicine,* agreed that the Library's usefulness should be increased but made a matter of record the not inconsiderable service presently being rendered. The article pointed out that greater community support must be offered if higher standards of performance are to be achieved. Attention was called to the fact that at the time of the writing of the articles, the College Library was not receiving support from municipal, state or federal government, other than tax status, and that no financial aid was being received from any county, state, or national medical societies.

Personnel. The Serials Department has earned special praise for maintaining high standards of service in the face of extensive personnel changes. Cecilia Chen Li, PH.D., the Head of the Department at the beginning of the report year, moved to New York with her husband at the end of his surgical residency in this area. Shortly after Mrs. Shi-Ping Show accepted the department headship, her husband was transferred out of the city. Mrs. Ping-Show remained on the staff through the summer until her duties were assumed by Miss Marybeth Honeyman in the fall. Miss Honeyman is a graduate of the School of Library Service of Villanova University and served in several departments in the Villanova University Library during her course of study.

Mrs. Doris Bridgman accepted a position in private business after a relatively short tour of duty in the Serials Department with primary responsibility for binding. This vacancy was filled by the appointment of Miss E. Naomi Frazer to the position of Serials Assistant. Miss Frazer has had extensive experience in special librarianship and editorial work. Miss Gayle

Donahower acquired her Drexel Library School degree and a husband at about the same time and left town with both. Mrs. Barbara Piper joined the staff in the Fall to cover the evening and Saturday schedule in the Periodicals Room. Miss Myrna Fisher also completed her studies at Drexel and accepted her first professional assignment in New York City. Mrs. Patricia Walsh accepted a library assistantship which divides her services between the Serials and Reference-Circulation Departments. Mrs. Paule Prebus also served in this Department until family responsibilities required her transfer to Columbus, Ohio.

Mrs. Rosayne P. Tumilowicz and Miss Judith A. Forquer served as readers' assistants while completing their studies at Drexel. After receiving their degrees, Mrs. Tumilowicz accepted a position at St. Agnes Hospital and Miss Forquer returned to her home city of Pittsburgh. These two part-time positions were filled by the appointment of Miss Janice K. Hill, who had been employed in the Fine Arts Library at the University of Pennsylvania. Mrs. Ferne Moffson served as an assistant in this department briefly before entering the teaching profession.

In the Processing Department, Mrs. Mercedes B. Sharpless filled the newly created position of Assistant Cataloger on a half-time basis for a short period of time. Her resignation was accepted with regret when her husband's transfer to Rutgers University took her out of town. This vacancy was combined with the vacancy created when Mrs. Theresa Perretta left the position of Cataloging Assistant to provide a full time Assistant Cataloger. Mr. Carmine D. Salvato, a graduate of the School of Library Service at Villanova University, was appointed to this new full time position.

Newcomers to the Paging Staff this year are Donna L. Pilson, Stephan Bilynsky, Judith Kimmel, and Veronica Enright. The following individuals, who are no longer on the Library staff, served as Library Pages during the year: Shirley Allen, Gloria Jean Culler, Terry Fox, Daniel Kelly, Constance Robinson, Linda Smith, and Brian E. Kleckner.

Professional Staff Activity. The Library was represented at the 65th Annual Meeting of the Medical Library Association in Boston by Mrs. Joyce Mingledorff, Miss Muriel Zeldis, and Elliott H. Morse. Mr. Morse participated in a panel discussion of the Resource Grant Program of the National Library of Medicine. He also continues to serve on the MLA/NLM Liaison Committee.

The Librarian participated as a speaker in an institute on Searching the Mental Health Literature held at the Eastern Pennsylvania Psychiatric Institute. He discussed the services offered by the National Library of Medicine. The Librarian also accepted an invitation to participate in the NLM Library Automation Workshop which was held in Bethesda early in November 1966. In March 1966 he served on the Oral Examining Board of the City of Philadelphia to review candidates for Library Assistant II.

Benefactors. A list of donors of books and other library materials appears in the statistical section of this report.

The Library acknowledges its gratitude to the following institutions which have shared the operational costs of the College Library:

NON-PROFIT INSTITUTIONS

Albert Einstein Medical Center, Medical Staff
Antioch College
Bryn Mawr Hospital Medical Staff
Chestnut Hill Hospital
Delaware Academy of Medicine
Fitzgerald-Mercy Hospital
Germantown Dispensary & Hospital
Hahnemann Medical College
Harrisburg Hospital
Jefferson Medical College & Hospital
Lankenau Hospital
Medical Literature, Inc.

Merck & Company, West Point
Misericordia Medical Staff
Philadelphia General Hospital
Temple University School of Medicine
U.S. Naval Hospital
University of Pennsylvania School of Medicine
University of Pittsburgh
Woman's Medical College of Pennsylvania

COMMERCIAL INSTITUTIONS

Air-Shields, Inc.
American Cyanamid Co. (Lederle Labs)
American Electronic Laboratories, Inc.
Atlas Chemical Company
James E. Beasley
Bell Telephone Company
Bernstein, Bernstein, Harrison & Kessler
Borden Chemical Company
Campbell Soup Company
Cohen & Verlin, Esq.
Council for Tobacco Research
Deichert, Price & Rhoads
Duane, Morris & Heckscher
E. I. du Pont de Nemours, Haskell Laboratory
E. I. du Pont de Nemours, Lavoisier Laboratory
E. I. du Pont de Nemours, Stine Laboratory
FMC Corporation (American Viscose Co.)
Frank C. Farnham Company
Feldman & Feldman
General Electric Co. Missile & Space Division
General Technical Services
Evelyn Goldstein
International Information Institute
Institute for Scientific Information
Kisselman, Devine, Deighan & Montana
LaWall & Harrison
Lewis & Gilman, Inc.
McNeil Laboratories
Mead Johnson & Company
William Miller
Mobil Oil Corporation
Morgan, Lewis & Bockius
Mullford Colloid Laboratories
Norwich Eaton Charitable Trust
Pennsalt Chemical Corporation
Pepper, Hamilton & Scheetz
Quaker Chemical Company
Radio Corporation of America
Ramsdell, Buckley & Company
Rohm & Haas Company
William H. Rorer, Inc.
Schering Corporation
Smith Kline & French Laboratories
Squibb Olin Company
Paula Stone
Sun Oil Company
Ted Thomas Associates

E. H. M.

Historical

Items Catalogued

Books added to the collection (including early works and current works on the history of medicine)	502
Pamphlets	700
Pictures, documents, and manuscripts	421
Books and pamphlets recatalogued (because of misleading cards, erroneous entries, or misleading location symbols)	104
	1,727

Cards Prepared

(These include 6,087 Xeroxed cards, which were headed, etc., by us; and 4,487 portrait cards, for individual portraits appearing in journals and books)	18,916

Special Activities

Housekeeping. With the major tasks of shifting and rearranging generally completed in the Cage, it has been possible to give increased attention to the care of individual volumes. Vellum bindings have been carefully washed (with surprising results), leather bindings have been oiled and softened with a special preservative ointment; acid-free paper slipcases have been made for small and medium-sized volumes in poor condition. The experience of our page, Miss Halina Wierzbiecki, in these and other preservative techniques has made it possible for the Associate Curator to spend a larger proportion of her time on "non-housekeeping" tasks.

Special Cataloguing Projects. 1) *"Fellows' cards."* The catalogue has now been brought up to date in respect to these cards, which bear each Fellow's full name, birth date, and year of election, and remain a permanent feature of our catalogue. Similarly, the small portrait photograph which each Fellow is asked to donate at the time of his election is preserved in the Picture File, and is recorded in our

Portrait Catalogue. This project has been the special task of Mrs. Betty Voigt, who has attended to it in the intervals of regular card production.

2) *Sturgis Collection cards.* The process has been begun of integrating the cards for the Sturgis Collection with those of the Portrait Catalogue (which lists individual portraits from books and journals, as well as engravings and photographs), and the Picture Catalogue (which lists artists, engravers, group portraits, places, buildings, objects, and so on). New cards are being made, based upon Dr. Sturgis's valuable descriptions; and location symbols are being added to cards and picture-mountings to insure return of borrowed materials to their proper portfolios. A considerable degree of industry applied to this project during the summer months has substantially completed about six of the more than 100 portfolios; this result reminds us once again of the magnitude of Dr. Sturgis's original contribution in collecting, preserving, mounting, and identifying these materials.

Sale of Historical Duplicates. Between January and October 31, 1966, a total sum of $962.50 was realized from the sale of duplicate books and pamphlets from the Historical Collections. Of this amount, $12.50 came from the sale of two items from List #1, and $270.00 from items chosen directly by their purchasers from "non-Listed" duplicates in the Historical Collections. The remainder, $680.00, was realized from List #2 of Duplicates, which contained 80 titles (some being plural copies), of which 34 have been sold.

L.M.H.

The conjunction of Mrs. Holloway's analytic cataloguing of one of the remarkable Joseph Carson Collections and the publication of Prof. Robert Brunhouse's *David Ramsay* provided a further striking demonstration of the loss to scholarship occasioned by the library's vagrant attention to historical cataloguing in past generations. In the course of his research on Ramsay Prof. Brunhouse visited the library, and his valuable monograph reproduces, or cites, several letters written by, or to, Ramsay that happened to have been catalogued; but the analytic cataloguing of the Carson Collection uncovered further letters that we had no key to at the time, and that would unquestionably have been of considerable value to him and the readers of his book.

Some interesting acquisitions:

1) Bally, François Victor (1775–1866). Du typhus d'Amérique ou fièvre jaune. Paris, 1814. (A prolific writer on epidemic diseases, Bally was for a time Physician-in-chief of the French army in Santo Domingo. The present work is said to be one of the best on this disease (yellow fever).)

2) Fosbroke, Thomas Dudley (1770–1842). Berkeley manuscripts . . . Biographical anecdotes of Dr. Jenner. London, 1821. (Title-page contains an ownership inscription: "Edwd. Jenner".)

3) Hirshberg, Leonard Keene (1877—). What you ought to know about your baby. A text book for mothers on the care and feeding of babies, with questions and answers especially prepared by the editor. New York, 1910. (Laid in was a typewritten, signed, letter (3 Dec. 1926) reading as follows: "Dear Mr. Dunlop: I find your note of November 3 on my return from California. The book you mention was a collaboration. Hirshberg supplied the materials and I put them into printable form. The chapters were first printed in the *Delineator,* then edited by Theodore Dreiser. The book was afterwards printed in a very large edition but it now seems to be somewhat rare. Sincerely, H. L. Mencken." Hirshberg received the M.D. degree from the Johns Hopkins University in 1902; thus, his as-

sociation with the famous writer, Mencken, could have begun in Baltimore and been continued in New York.)

4) Nannoni, Angelo (1715–1790). (a) Trattato chirurgico sopra la semplicità del medicare i mali d'attenenze della chirurgia. Venice, 1764; (b) Trattato chirurgico sopra la semplicità del medicare i mali curabili coll'ajuto della mano. Contenente, le memorie, e osservazioni, che molto giovano per lo stabilimento del buon metodo di medicare i mali appartenenti al chirurgo. Siena, 1774. (Two editions, by no means identical, of an important surgical text by one of the most celebrated of Italian surgeons.)

5) Petrini, Giuseppe (1760–). Nuovo metodo di guarire la sciatica nervosa. Rome, 1781. (Petrini's teacher, Cotugno, is credited with a classical description of sciatica; the pupil here describes different methods of treating the disease.)

6) Pettenkofer, Max von (1818–1901). Zum gegenwärtigen Stand der Cholerafrage. Munich, 1887. (One of the author's most important publications.)

7) Spratt, George. Obstetric tables: comprising graphic illustrations, with descriptions and practical remarks; exhibiting on dissected plates many important subjects in midwifery. 3d ed. 2 pts. London, 1835. (The library lacked an English imprint of this popular work, the plates of which are of especial interest for their use of the device of superimposed flaps, most frequently associated with the early anatomical fugitive sheets.)

Exhibits

In addition to exhibits based on the programs of the Section on Medical History, an appropriate exhibit was prepared for the annual meeting of the Halsted Society, at the request of the Bicentennial Committee of the University of Pennsylvania; this was followed by an exhibit designed to be of interest to those attending the Clinical Meeting of the American Medical Association. Another exhibit —possibly verging on the "far out" in concept—displayed notable works by writers who either were born or died in the 16th or 66th year of the 16th–19th centuries, the present century being represented by its 16th year only. Offering a cross-section of the library's historical texts, the 20 volumes shown—in, variously, Latin, French, Italian, and English —drew on writers from the following countries; Austria, Denmark, England, France, Germany, Italy, Russia, Switzerland, and, of course, the U.S.A. The College's Herb Garden was visited in May by a large group of persons attending the annual meeting of the Herb Society of America. At the request, and with the valuable assistance, of Mrs. Henry Cadwalader, a large exhibit of early printed herbals in the library was on display. As a means of easy reference, it included a copy of the issue of the *Fugitive Leaves* (April, 1962) that constitutes a check-list of our collection. To our surprise, the result was a number of orders for Xerox copies of the issue, though the check-list had been published almost simultaneously in *The Herbarist,* the Society's journal. The final major exhibit, on pulse-lore, drew largely on the catalogue of a similar exhibit that had been on view during the summer at the Royal College of Physicians, in London. The library's historical collections were appropriately represented in the initial historical exhibit at the new William Penn Museum, in Harrisburg, by the loan of classic works from the pens of John Morgan, John Jones, and Nathaniel Chapman, noted Fellows of the College who were pioneers in the field of American academic medical education.

Fugitive Leaves. There were eight issues of the *Leaves* during the report-year, two more than in the report-year immediately preceding. They (N.s., nos 79–86) were all

devoted to reproduction of John Morgan material in one of the Carson Collections mentioned at the beginning of this section of the report.

Pictorial Collections. As has been the case in recent years, this material has been drawn on continuously and heavily for reproduction. A two-day visit, for instance, from two staff members of one of our leading pharmaceutical houses resulted in an order for photographic negatives of 21 illustrations in 10 volumes, ranging from the 16th to the 20th centuries. Then, one of the library's all-but-unique set of colored plates in a 17th c. edition of Fabricius ab Aquapendente's *De formato foetu* was reproduced in color on the cover of the Oct. 10, (1966) issue of the *Journal of the American Medical Association.*

Curatorial publications. In addition to selecting and editing the material published in the *Fugitive Leaves,* mentioned above, the senior Curator was represented in the April, 1966, issue of the College's *Transactions & Studies* by two brief historical communications; and in the Fall 1965 issue of *Isis* by a book-review.

Pax vobiscum: Edward Bell Krumbhaar, M.D., 1882–1966—President of the College (1940–42), eminent benefactor, scientist, scholar, loyal friend.

W. B. McD., 2d

Medical Documentation Service

During the report year ending October 31, 1966 the Medical Documentation Service reported to its subscribers nearly 7,000 references. The average number of subjects was 29. Comparable figures for 1965 (hereafter given in parentheses after the current data) was 4574 references and 26 subjects. This increase in work done is further indicated by the income from scanning which was $15,709. ($10,268.).

We prepared 1575 (775) abstracts and the income of $20,400. ($16,216.) did not parallel the increase.

The number of literature searches was 208 (276) and the income was $3,728. ($5,518.). In accordance with a policy initiated during the year, Fellows of the College received up to five hours' free service in the preparation of bibliographies. Nine Fellows had 12 searches made which took 24 hours of professional time. No publicity has been given this since we have only limited staff time available.

The income from translations is a clear indicator of work done. It increased to $10,136. ($9,211.).

The staff of three full-time employees has continued to render cheerful service. Jean Carr is the keystone of the arch as the Executive Secretary. Barbara Flood resigned early in 1966 and J. Louise Walsh worked as abstracter-translator during much of the year under review. She moved to Washington, D. C. in July and Mrs. Marianne Das, formerly of Biological Abstracts and known to all of us for several years, became our staff abstracter-translator. These competent and loyal employees were supplemented and complemented by Patricia E. Roddy, now in her senior year at Temple University, Gail E. Groeneman, our copy editor, Arlette Sherman, a part-time typist, Elizabeth Bowman Wright, typist and others. Our freelance translators and abstractors also make a valued contribution to our work.

Eleanor Ashkenaz, Ph.D., of West Chester State College, was acting director during July 1966 while the director had a month's vacation. He is grateful to her and to the Librarian for making this possible.

The fiscal year ending June 30, 1966 showed an excess of income over expenditures if our customary accounting procedures had been applied. However, if the Service were charged for its fair share of College operating costs, it is likely that a deficit would have resulted.

W.L.B.

STATISTICAL SUMMARY

Inventory

Accessioned Items:[1]

	1966		*1965*	
Incunabula[2]	421		421	
Manuscripts and typescripts	1,300		1,294	
General	231,755		227,061	
Accessioned pamphlets	7,407		6,688	
		240,883[3]		235,464[3]
Unaccessioned Items:[1]				
Reports	28,815		28,814	
Theses	62,184		61,871	
Pamphlets and reprints	228,492		229,396	
		319,491[4]		320,081

Accessioned Acquisitions:

1966: 5,419 volumes (2,374 by purchase; 2,604 by gift; 441 by exchange). (Books: 1,750; Periodicals: 2,950; Pamphlets: 719).

1965: 5,802 volumes (2,692 by purchase; 2,603 by gift; 507 by exchange). (Books: 1,600; Periodicals: 3,550; Pamphlets: 652).

Non-accessioned acquisitions:

1966: 345 items (Reports, 1; Theses, 313; Miscellaneous, 31).

1965: 468 items (Reports, 8; Theses, 435; Miscellaneous, 25).

Currently Received Serials

	Subscription	*Gift*	*Exchange*	*Total*
U.S.A.	495	456	59	1010
Western European Continent	761	83	137	981
Eastern European Continent	159	57	61	277
United Kingdom and Eire	144	36	30	210
Asia (Including Japan)	80	89	82	251
Latin America and West Indies	52	67	107	226
International	93	12	1	106
Canada	17	19	6	42
Pacific (Including Australasia)	6	14	10	30
Africa	4	9	15	28
United Nations	10	—	—	10
Grand Total 1966	1821	842	508	3171[5]
Grand Total 1965	1776	931	503	3210

New Titles: A total of 81 new serial titles was added in 1965/66.

Separate issues received, checked in the Kardex record and shelved totaled 24,526.

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[1] Accessioned items in this library comprise the total number of bound volumes and such unbound pamphlets as have been cataloged as separate bibliographical units regardless of their size.

There remain many thousands of unaccessioned pamphlets awaiting accessioning and cataloging. A true quantitative and qualitative picture of the library's holdings cannot be presented until this wealth of neglected material has been processed and duly registered in our 'accessioned' count.

[2] The Library owns 423 incunabula, but there are two cases of two bound together, reducing the number of accessioned items.

[3] Included in these figures are the following collections which are on permanent deposit in the Library: Gross Library, 3,976 accessioned items; Parry Library of the Obstetrical Society of Philadelphia, 217; Mütter Museum, 200 (Mütter Museum books may not be removed from the College building).

[4] Decline in this figure results from weeding project described in last year's report.

Readers' Use of the Library

Library hours. The Library was open, during the academic year, from 9:00 to 9:30 on Mondays and Wednesdays, from 9:00 to 5:00 on other weekdays. During June, July, August, and until the first Monday after Labor Day, the Library was open from 1:00 to 9:00 on Tuesdays and from 9:00 to 5:00 on other weekdays, except Saturdays, when it was closed. The following legal holidays were observed: Thanksgiving, Christmas, New Year's, Memorial, Independence, and Labor Days.

Visitors:

	Reading Room	*Periodicals Room*	*Totals*
1966	9,213	3,409	12,622
1965	10,240	3,392	13,632

Circulation:

	Intra-Mural[6]	*Extra-Mural*	*Totals*
1966	33,317	39,158	72,475
1965	34,325	33,025	67,350
	Books	*Journals*	*Totals*
1966	12,282	60,193[7]	72,475
1965	12,153	55,197[8]	67,350

Interlibrary loans: The Library sent 27,359 (1965: 21,736) volumes on interlibrary loan to 300 (1965: 338) libraries in 33 (1965: 32) states, Canadian provinces and foreign countries.

Photoduplications: 139,816 pages were photocopied on request (1965: 109,971).

Donations

The donors for the past year number 85 (1965: 85). The Library is indebted for large gifts of books, pamphlets and unbound periodicals to the following donors: American College of Physicians; Dr. S. Bellet; Biological Abstracts; Drs. S. Ellis, R. J. Gould; Grand View Hospital; Hahnemann Medical College; Hospital of the University of Pennsylvania; Dr. S. L. Israel; Jefferson Medical College; Drs. M. D. Kasser, R. A. Kern; Lea & Febiger; Drs. E. R. Long; C. Macfarlane; Mr. J. Mach; Merck Sharp & Dohme; Drs. S. Mudd, A. A. Nardone; National Foundation; Penn Mutual Life Insurance Co.; Presbyterian-University of Pennsylvania Medical Center; Dr. M. Scarf; Smith Kline & French; Wallace Laboratories; Dr. I. J. Wolman; Woman's Medical College.

Various publishing houses, among the large group of corporate donors, have presented volumes as follows: F. A. Davis Co., 44; Lea & Febiger, 17; J. B. Lippincott Co., 72; W. B. Saunders Co., 95.

Other individual donors were as follows: Drs. H. J. Abrahams, R. Asnis, D. M. Aviado, C. G. Barber, O. V. Batson, W. J. Burdette; Mr. J. R. Burke; Drs. J. E. Eckenhoff, M. Finkelstein, P. S. Friedman, B. D. Gettes, W. K. Hall, E. H. Hinman; Mr. E. R. F. Johnson; Drs. L. C. McHenry, Jr., S. Mudd, Mr. E. J. Neren; Miss M. G. Palmer; Drs. L. C. Parish, F. E. Proctor, S. X Radbill, T. K. Rathmell, S. P. Reimann, S. Rubin, J. B. Rudolphy, I. S. Tassman; Miss E. Westcott; Drs. J. B. Wolffe, R. J. Wolfson.

The Transactions & Studies was sent, either in exchange or as gift, to 547 organizations.

Theses and dissertations to the number of 339 have been received on exchange from the following European schools of medicine: Universities of Basel, Bern, Erlangen, Geneva, Karolinska Institute of Stockholm, Leiden, Liège, Louvain, Lund, Uppsala, and Utrecht.

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[5] Titles are only included in this count if issues were received in 1965 or 1966. The decline in this year's count reflects extensive culling of inactive and out of scope titles.

[6] The number of volumes "consulted in the library" includes only those supplied on demand. Readers have access to the bound volumes of periodicals and reference works kept on the shelves in the Reading Room; the Fellows, and occasionally others, by special permission, have access to the book stacks. There are, therefore, many volumes consulted of which no accurate record can be kept.

[7] This figure includes 6,566 unbound current journal issues circulated from the Periodicals Room.

[8] This figure includes 4,853 unbound current journal issues circulated from the Periodicals Room.

The Staff[9]

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Library Secretary
Mildred C. Gray

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Muriel Zeldis, *Head*

Cataloger
Carmine Salvato

Assistant Cataloger
Mrs. Kathryn E. Miragliotta

Reference-Circulation Dept.

Mrs. Joyce S. Mingledorff, *Head*

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Mrs. Julia O'Malley
Mrs. Kathryn Bernice Odom*
Janice K. Hill

Serials Dept.

Marybeth Honeyman, *Head*

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Mrs. Catherine Ledwell
E. Naomi Frazer
Barbara D. Maurer
Patricia Walsh
Mrs. Barbara Piper*

Historical Collections

W. B. McDaniel, 2d, Ph.D., *Curator*
Mrs. Lisabeth M. Holloway, *Associate Curator and Historical Cataloger*
Mrs. Betty Voigt,* Typist

Medical Documentation Service

Walter L. Bethel, Ph.D., *Director*
Jean P. Carr, *Executive Secretary*
Marianne Das, *Abstracter/Translator*
Mrs. Gail E. Groeneman* *Copy Editor*
Mrs. Elizabeth Wright* *Typist*
Ruth I. Weidner* *Copy Editor*
Robert B. Blackerby* *Copy Editor*

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Josefa Sereda
Halina T. Wierzbicki*

Approved:
Samuel X Radbill, M.D.
Chairman, Library Committee
As of February 15, 1967

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[9] Library Staff list as of February 15, 1967.
* Part-time

Annual Report of Committee on the Mütter Museum and College Collections

If former visitors to the Mütter Museum of The College of Physicans made a return visit this year and were at all observing, they would have noticed that a new ceiling and new lighting have been installed by the superintendent, adding to the appearance of the rooms and giving much greater illumination. Some of the floors have also been given a fresh coat of paint.

A rearrangement of the large microscope collection has been made. It was removed from the crowded position in one of the old museum cases and transferred to one of the attractive ones given to the museum on a permanent loan by the Wistar Institute.

Most of the cases and specimens have received their regular cleaning and other attention for 1966, and, because most of the old cases are not dust proof and most of the jars not air tight, cleaning cases and replenishing formalin will soon begin again for 1967. These are continuous chores in the museum.

This year the usual classes from the Woman's Medical College and Hahnemann Medical College, who have been coming regularly for a number of years, were unable to attend. Their absence, however, did not reduce the attendance, which numbered 2452, as much as one would suppose since there was an increase in the individual attendance and there were new groups who arranged visits. One of these groups was students from the Manpower Training School in the city. They were receiving instructions in medical typing and were anxious to associate medical terminology with actual specimens.

A biology class of thirty from the Medford High School in Medford, New Jersey, came with Mr. John Jones, their instructor, and as the entire class numbered over 100, arrangements were made for the rest of the class to come to the museum in small groups in January, February and March.

More interest in the museum collection was shown by students of nursing this year. Montgomery Hospital in Norristown has been sending small groups each Tuesday during the year. Student nurses also came from Fitzgerald Mercy, Cherry Hill and Lankenau Hospitals.

The Franklin School of Science and Arts is located near the College, so many of the students of Medical Technology come to the museum. We can expect some of them to drop in almost every day.

Beside the biology class from Medford High School, Central, Clifton Heights and Roxborough High Schools sent students in large numbers. Students from the Moravian Prep School, which is connected with the Moravian College in Bethlehem, Pennsylvania, made a surprise visit.

Students from St. Leonard's Academy, under the able leadership of Sister Adelaide, are ones we welcome gladly. These girls are personable, intelligent and well-behaved. Knowing Sister Adelaide, who darts from one group to another, pointing out specimens they might have missed, one can understand how their interest and deportment can be attributed to an enthusiastic and dedicated teacher.

Medical students from Temple University, accompanied by Dr. Fred B. Rogers, made a tour of the library and museum. Jefferson Medical College students also found a visit rewarding. Another group came from the Temple University School of Pharmacy.

During the holidays Mr. George Purvis brought a small group of students from the Plymouth-Whitemarsh Senior High School.

As in past years, our register shows the names of many graduate medical students from foreign countries.

The museum is open from 9 a.m. to 3:30 p.m., Monday through Friday, and special arrangements are made for groups who cannot come at the stated time.

The 78th Thomas Dent Mütter Lecture was delivered on February 2, 1966 by Averill A. Liebow, M.D., Professor of Pathology, Yale University School of Medicine, New Haven, Connecticut, his topic being *Hypersensitivity Reactions Involving the Lung.*

The 79th Mütter Lecture was given on November 2, 1966 by Donald G. McKay, M.D., Professor and Chairman of Pathology, College of Physicians and Surgeons of Columbia University, New York. His lecture was entitled *Endotoxin Shock.*

The museum furnished copies of the partial catalogue of the Hyrtl and Politzer ear collection to George T. Nager, M.D., from Johns Hopkins Hospital. Photographs of the Spencer-Wells pedicle clamps in the collection were sent to Dr. K. A. Shepherd of London, England. He intends to use them to illustrate a paper he is writing.

Periodically, special interest is shown in the tumor removed from President Cleveland's jaw in 1893. This is in a case at the entrance to the museum, and recently requests were made by several physicians for permission to take a small piece from the specimen for biopsy. Permission was refused by the Committee on the Mütter Museum and Council of the College. Dr. William Biederman of New York was permitted to photograph the laryngeal mirror and retractor used in the operation.

Dr. Samuel X. Radbill's class in pediatrics was given a tour of the museum with special attention called to those exhibits in which they would be especially interested.

There are no accessions to report for the museum as all gifts were accepted for College Collections where they are available for loan.

Mrs. Ella N. Wade was reappointed curator for the year 1966–67.

College Collections

The Committee on College Collections lent the watch of Dr. David Hayes Agnew which he wore when in attendance on President Garfield, and also the cane carried by Dr. Benjamin Rush, to the William Penn Memorial Museum in Harrisburg, for a special exhibit held from April 28th to August 31st, 1966. The committee also permitted Mr. Irwin Richman of the William Penn Museum to have a photograph of the minature of Dr. Thomas Bond for a permanent exhibit.

During the years from 1942 to 1960 Dr. Edward F. Corson presented a large collection of British War Medals to the College Collection. Some time ago he suggested they be sold because of an apparent lack of interest in them, and the money used for some other purpose. This met with the approval of Council. His offer to find a buyer, however, did not materialize and it was the opinion of the committee that instead of selling them, as the museum was also unable to find a market, it would be a nice gesture to donate them to an institution that would be interested in them. We were unsuccessful in finding anyone who wanted them until April of this year, when Dr. and Mrs. Cyril Adler visited the museum. Dr. Adler is Honorary Director of the Museum of Medical History at the University of Witwatersrand, Johannesburg, South Africa, and Mrs. Adler is the Honorary Curator. They were shown the medals and were delighted to accept them, as donated by the College, with special mention made of Dr. Edward F. Corson as the original donor.

Mr. Sydney Thayer, Jr., of Bryn Mawr, a great-great grandson of Dr. Nathaniel Chapman, donated a silver pitcher that was presented to Dr. Chapman in 1833 by the City of Philadelphia, for the conspicuous part he had taken in treating the sick during an epidemic of Asiatic cholera. Twelve other physicians were similarly honored. A short article about the pitcher appeared in the October 1966 issue of the *Transactions & Studies* of the College, and the pitcher is temporarily displayed in a section of the medical medal case in the second floor hallway.

Dr. Nicholas Padis presented a small surgical instrument from Epidaurus, Greece, and *Dr. Fred B. Rogers* presented a 1907 Bausch & Lomb microscope that formerly belonged to his father, Dr. Lawrence H. Rogers of Trenton, New Jersey. *Dr. George I. Blumstein* donated a case of surgical instruments that were used during the Civil War.

Mrs. Ella N. Wade was reappointed custodian of College Collections for the year 1966–67.

Nicholas Padis, M.D.
Chairman

List of College Lectures, 1966

5 January 1966

COHEN, SEYMOUR S. Virus-Induced Synthesis of Enzymes (Nathan Lewis Hatfield Lecture L; *Transactions & Studies,* April 1966)

2 February 1966

LIEBOW, AVERILL A. Hypersensitivity Reactions Involving the Lung (Thomas Dent Mütter Lecture LXXVIII; *Transactions & Studies,* October 1966)

2 March 1966

MASTROIANNI, LUIGI, JR. Control of Conception in Primates (James M. Anders Lecture XLIV; *Transactions & Studies,* July 1966)

6 April 1966

WOODWARD, THEODORE E. An American Clinician in Asia (Benjamin Musser Lecture IX; *Transactions & Studies,* July 1966)

4 May 1966

MCEWEN, CURRIER. Reiter's Disease: Its Nature and Relationship to Other Diseases (Theodore F. Bach Memorial Lecture I; *Transactions & Studies,* October 1966)

18 May 1966

Joint Meeting of the Philadelphia County Medical Society and The College of Physicians of Philadelphia.

I. Presentation of the Second Annual Kenneth E. Appel, M.D., Award to Melvin Singer, M.D., Institute of the Pennsylvania Hospital.

II. Presentation of Certificates to Physicians who have completed fifty years in the practice of medicine.

III. Presentation of the Strittmatter Award to Eugene P. Pendergrass, M.D., Emeritus Professor of Radiology, University of Pennsylvania School of Medicine.

5 October 1966

SCRIVER, CHARLES R. Cellular Transport of Amino Acids in Man (Mary Scott Newbold Lecture LXXXXI)

2 November 1966

MCKAY, DONALD G. Endotoxin Shock (Thomas Dent Mütter Lecture LXXIX; *Transactions & Studies,* April 1967). A Combined Meeting of The Philadelphia County Medical Society, The Pathological Society of Philadelphia and The College of Physicians of Philadelphia.

7 December 1966

POPPER, HANS. Organelle Pathology of the Liver (Alvarenga Prize Lecture XXVI; *Transactions & Studies,* April 1967)

Transactions of the Sections

SECTION ON MEDICAL HISTORY

8 February 1966

CLASSIC DESCRIPTION IN DIAGNOSTIC ROENTGENOLOGY: THE HISTORY OF DARK ADAPTATION. William J. Tuddenham, M.D.

THE EARLY DAYS OF OPHTHALMOLOGY AT THE UNIVERSITY OF PENNSYLVANIA. Daniel M. Albert, M.D., and Harold G. Scheie, M.D.

CONTEMPORARY HISTORY AND BIOGRAPHY IN PSYCHIATRY: DANIEL BLAIN, M.D., Kenneth E. Appel, M.D.

26 April 1966

JAMES HUTCHINSON (1752–1793): LETTERS FROM AN AMERICAN STUDENT IN LONDON. Whitfield J. Bell, Jr., PH.D. (The Kate Hurd Mead Lecture)

11 October 1966

W. SOMERSET MAUGHAM (1874–1965): A MEDICAL TRUANT. Fred B. Rogers, M.D.

THE MEDICAL HISTORY AND THE TYPE OF INJURIES SUSTAINED BY THE FOUR PRESIDENTS OF THE UNITED STATES WHO WERE ASSASSINATED. T. A. Ranieri, M.D.

THE ALBERT EINSTEIN MEDICAL CENTER OF PHILADELPHIA: FIRST 100 YEARS. J. Gershon-Cohen, M.D.

18 November 1966

G. C. KRATZENSTEIN—A GREAT PERSONALITY AMONG DANISH PHYSICIANS IN THE LATTER PART OF THE 18th CENTURY. Egill Snorrason, M.D.*, Secretary, Danish Society for the History of Medicine.

SECTION ON OPHTHALMOLOGY[1]

3 February 1966

THE IDEAL TECHNIQUES OF CATARACT SURGERY. Panelists: P. Robb McDonald, M.D., James S. Jordan, M.D.*, Arthur H. Keeney,* M.D. Discussants: Charles G. Steinmetz,* M.D., Bernard C. Gettes, M.D.

17 March 1966

DIABETIC RETINOPATHY. Myron Yanoff, M.D.*

INNERVATION OF THE CENTRAL RETINAL ARTERY: DEMONSTRATION BY HISTOFLUOROMETRIC TECHNIQUE. Alan M. Laties, M.D.*

ORAL GLYCEROL: MODE OF ACTION. Bernard Schneider, M.D.*, Donna K. McCurdy, M.D.*, and Harold G. Scheie, M.D.

HEMOPHILIA AND THE EYE. Robert A. Rubenstein, M.D.*, Daniel M. Albert, M.D.*, and Harold G. Scheie, M.D.

21 April 1966

CORNEAL ULCERS IN KERATITIS SICCA. William C. Frayer, M.D. and J. G. Gleichaur, M.D.*

FLUORESCEIN STUDIES OF THE FUNDUS. Thomas Behrendt, M.D.* and Edward A. Jaeger, M.D.*

RADIATION OF METASTATIC INTRAOCULAR TUMORS. Simon Kramer, M.D.* and Thomas D. Duane, M.D.

RUBELLA RETINITIS. J. C. Toland, M.D.*

27 October 1966

PROBLEMS OF ENUCLEATION. David B. Soll, M.D.

OCULO-AURICULO-VERTEBRAL DYSPLASIA. Robert Kiessel, M.D.* and R. D. Harley, M.D.*

HISTOPATHOLOGY OF CORNEAL GRAFT FAILURES. George H. Kurz, M.D.

17 November 1966

THE MANAGEMENT OF FUNGUS KERATITIS. Arthur Gerard DeVoe, M.D.*, Professor of Ophthalmology, College of Physicians

* By invitation.

[1] Abstracts of the presentations at this Section customarily appear in the *American Journal of Ophthalmology* and *A.M.A. Archives of Ophthalmology*.

and Surgeons, Columbia University, New York. (Twenty-Ninth Annual deSchweinitz Lecture)

SECTION ON OTOLARYNGOLOGY[2]

5 April 1966

(Combined meeting, Section on Otolaryngology, The College of Physicians of Philadelphia and the Philadelphia Laryngological Society.) Resident Program:

GIANT CELL TUMOR OF THE NOSE. Harvey Yeager, M.D.

SEROUS OTITIS MEDIA MEDICAL TREATMENT. Myles G. Turtz, M.D.

MEGALO-TRACHEA AND BRONCHIAL TREE. Eurico Almeida, M.D.

[2] Abstracts of the presentations at the Section customarily appear in the *A.M.A. Archives of Otolaryngology*.

EXPERIMENTAL STAPEDECTOMY WITHOUT OVAL WINDOW COVER. Gary K. Thomas M.D.

DERMOID CYST OF FLOOR OF THE MOUTH. Javad Sholehvar, M.D.

SECTION ON PUBLIC HEALTH, PREVENTIVE AND INDUSTRIAL MEDICINE

17 November 1966

PREVENTIVE APPROACH TO COMMUNITY PRACTICE. George T. Harrell, M.D.*, Dean, The Pennsylvania State University College of Medicine, and Director, The Milton S. Hershey Medical Center, Hershey, Pennsylvania. (Arthur Parker Hitchens Lecture XI. Joint meeting with Pennsylvania Public Health Association, Region One.)

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To serve until January, 1969

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Mütter Museum and College Collections

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* The President is an *ex officio* member of all Standing Committees.

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February 1, 1967

ELECTED

1952. Abelson, Neva Martin, 32 Woodale Rd. (18)
1961. Adamson, William C., 1542 Susquehanna Rd., Rydal, Pa.
1926. Adler, Francis Heed, 313 S. 17th St. (3)
1959. Adlin, Albert, 1512 Lindley Ave. (41)
1949. Aegerter, Ernest E., 3400 N. Broad St. (40)
1953. Agerty, Horst A., 1111 N. 63rd St. (19151)
1960. Ainsworth, Thomas H., Jr., 960 County Line Rd., Bryn Mawr, Pa.
1964. Albert, Seymour M., 255 S. 17th St. (19103)
1963. Albright, Edwin P., 512 E. Mt. Airy Ave. (18)
1967. Alexander, Charles M., 930 W. Sproul Road, Springfield, Pa. (19064)
1958. Alexander, John Deaver, Philadelphia General Hospital (4)
1962. Alexander, John Dewey, Jr., 330 S. 9th St. (7)
1935. Alpers, Bernard J., 111 North 49th St. (39)
1960. Ames, Mary D., 260 St. James Place (6)
1961. Amsterdam, Gerald H., Wyncote House, Wyncote, Pa.
1962. Annesley, William H., Jr., 39 Glendale Road, Upper Darby, Pa.
1961. Anthony, Russell A., 385 Lancaster Ave., Haverford, Pa.
1961. Aponte, Gonzalo E., 1025 Walnut St. (7)
1930. *Appel, Kenneth E., 111 N. 49th St. (39)
1905. Appelman, Leighton Francis, Limekiln Pike & Twining Rd., Glenside, Pa. (19038)
1960. Arey, James B., 2600 N. Lawrence St. (33)
1959. Arkless, Henry A., 255 S. 17th St. (3)
1922. Arnett, John H., 2116 Pine St. (3)
1952. Ash, Rachel, 255 S. 17th St. (3)
1952. Aston, Melville J., 6 Circle Drive, Margate, N.J. (08402)
1944. Atkins, Joseph P., 1201 Knox Rd., Wynnewood, Pa.
1956. Atkinson, John B., 410 Township Line Rd., Havertown, Pa. (19083)
1935. Atlee, John L., Jr., 37 E. Orange St., Lancaster, Pa.
1952. Atlee, William A., 37 E. Orange St., Lancaster, Pa.
1963. Austrian, Robert, Hospital of the University of Pennsyvania (4)
1958. Ayella, Alfred S., Jr., 1213 S. Broad St. (47)
1960. Bacon, Catherine L., 504 Howe Rd., Merion Station, Pa.
1937. Bacon, Emily P., 803 E. Darby Rd., Havertown, Pa. (19083)
1958. Baer, Samuel, 5123 N. Broad St. (41)
1959. Baird, Henry Welles, III, 2600 N. Lawrence St. (33)
1952. Baier, Howard N., 1333 Chestnut St. (19107)
1952. Baker, Howard W., 3401 N. Broad St. (40)
1963. Baker, Richard B., 1851 E. Allegheny Ave. (34)
1946. Baker, Walter W., 255 S. 17th St. (3)
1963. *Balin, Howard, 330 S. 9th St. (7)
1957. Baltzell, William Hewson, 1530 Locust St. (2)
1940. Barba, Philip S., 120 Erdenheim Road (18)
1943. Barden, Robert P., 8835 Germantown Ave. (18)
1939. *Barnshaw, Harold D., 526 Cooper St., Camden 2, N. J.
1957. *Barr, Stanley, 255 S. 17th St. (3)
1939. Bartle, Harvey, Jr., Glenbrook Medical Bldg., 864 County Line Rd., Bryn Mawr, Pa.
1963. Bassett, James G., 3300 Henry Ave. (19129)
1932. Batson, Oscar V., 3926 Pine St. (4)
1966. Baue, Arthur E., Graduate Hospital (19146)

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1921. Bauer, Edward L., Alden Park Manor (44)
1933. Bauer, John T., 312 E. Second St., Moorestown N. J.
1939. Baumann, Frieda, 200 N. Wynnewood Ave., Wynnewood, Pa.
1930. Beardwood, Joseph T., Jr., 1245 Highland Ave., Abington, Pa. (19001)
1952. *Bedrossian, E. Howard, 4501 State Rd., Drexel Hill, Pa.
1935. Beerman, Herman, 255 S. 17th St. (3)
1942. Behrend, Albert, Coventry House, Valley Rd. & Coventry Ave. (19126)
1945. Behrend, Bernard, 5910 Greene St. (44)
1934. *Behrend, Moses, Rittenhouse Claridge, 18th & Walnut Sts. (3)
1949. Beizer, Lawrence H., 419 South 19th St. (46)
1936. Bell, Benjamin Tertius, 1941 Woodland Rd., Abington, Pa.
1956. Beller, Martin Leonard, 1936 Spruce St. (3)
1947. Bellet, Samuel, 2021 Spruce St. (3)
1956. Belmont, Owen, 5723 No. Park Ave. (41)
1959. Berk, Nathaniel G., 2534 W. Cheltenham Ave. (50)
1951. Bernstine, J. Bernard, 255 S. 17th St. (3)
1965. Berry, Richard G., 1025 Walnut St. (19107)
1956. Berry, Theodore J., 331 E. Lancaster Ave., Wynnewood, Pa.
1957. Biele, Albert M., 1530 Locust St. (2)
1964. Biele, Flora H., 1530 Locust St. (19102)
1918. Billings, Arthur E., 2020 Spruce St. (3)
1957. Birdsall, Thomas M., 255 S. 17th St. (3)
1949. Bishop, Edward H., 811 Spruce St. (7)
1933. Bishop, Paul A., 1347 Waverly Rd., Gladwyne, Pa.
1944. Blady, John V., Parkway House, 2201 Benjamin Franklin Pkwy. (30)
1960. Blake, Alton D., Jr., Bryn Mawr Medical Bldg., Bryn Mawr, Pa.
1952. Blake, Paul O., 273 N. Lansdowne Ave., Lansdowne, Pa.
1958. Blakemore, William S., 419 S. 19th St. (46)
1917. Block, Frank B., A1124 Park Drive Manor (44)
1963. Block, Reuben, 1555 Haddon Ave., Camden, N. J.
1949. *Blumstein, George I., 2039 Delancey St. (3)
1929. *Bockus, Henry L., 250 S. 18th St. (3)
1962. Boggs, Thomas R., Jr., Children's Hospital, 1740 Bainbridge St. (46)
1928. *Boles, Russell S., Rittenhouse Plaza, 1901 Walnut St. (3)
1923. Bond, Earl D., Bryn Mawr Court, Bryn Mawr, Pa.
1961. Bongiovanni, Alfred M., 1740 Bainbridge St. (46)
1948. Bookhammer, Robert S., 264 Forrest Rd., Merion, Pa.
1931. Bortz, Edward Le Roy, Lankenau Medical Bldg. (19151)
1936. Bostwick, Delazon Swift, 1523 Sweetbriar Rd., Gladwyne, Pa.
1947. Bourland, Henry S., 2219 Garret Rd., Drexel Hill, Pa.
1961. Bouzarth, William F., 3300 Henry Ave. (29)
1961. Bove, Frank A., 2301 Pennsylvania Ave. (30)
1954. Bowers, Paul A., 2031 Locust St. (3)
1942. Bowie, Morris A., Bryn Mawr Medical Bldg., Bryn Mawr, Pa.
1963. Boyer, Randal A., Holy Redeemer Hospital, Meadowbrook, Pa.
1964. Brackin, John T., Jr., Lansdale Clinic Bldg., S. Broad St. & Allentown Rd., Lansdale, Pa. (19446)
1965. Brady, Anna M., 3302 W. Queen Lane (19129)
1959. Brady, Luther W., 230 N. Broad St. (19102)
1954. Bralow, S. Philip, 3701 N. Broad St. (40)
1942. Branen, William T., Whitby & Cypress Aves., Yeadon, Pa.
1921. Bransfield, John W., 2031 Locust St. (3)
1948. Braun, William, 406 Cooper St., Camden, N. J.
1953. *Brav, Solomon S., 5575 N. Park Ave. (41)
1953. Breckenridge, Robert L., Our Lady of Lourdes Hosp., 1600 Haddon Ave., Camden 3, N. J.

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* S. Weir Mitchell Associate.

1964. Brenman, Arnold K., 3701 N. Broad St. (19140)
1966. Brent, Robert L., Jefferson Medical College (19107)
1961. Breslow, Irwin H., 1924 Panama St. (19103)
1946. *Briscoe, Clarence Conway, 811 Spruce St. (7)
1953. Brobeck, John R., Univ. of Pa. School of Medicine (4)
1958. Brody, Henry, Einstein Medical Center, York & Tabor Rds. (41)
1965. Brody, Jerome I., Graduate Hospital, 19th & Lombard Sts. (19146)
1952. Brody, Morris W., 2 W. Levering Mill Rd., Cynwyd, Pa. (19004)
1948. Brogan, Edmund J., 1020 Cedargrove Rd., Wynnewood, Pa.
1958. Brooks, Frank P., Hospital of the Univ. of Pa. (4)
1936. *Brown, Clark E., Lancaster Ave. & City Line Ave. (19151)
1932. *Brown, Claude P., 1930 Chestnut St. (3)
1963. Browne, Laurence T., Presidential Apts., D126 (19131)
1961. Brunt, Manly Y., 111 N. 49th St. (39)
1939. Brust, Raymond M., 301 Mill Rd., Havertown, Pa. (19083)
1953. Bucher, Robert M., 3401 N. Broad St. (40)
1961. Buerk, Minerva S., Bryn Mawr Medical Bldg., Bryn Mawr, Pa.
1962. *Burgoon, Carroll F., Jr., Skin & Cancer Hospital, 3322 N. Broad St. (19140)
1940. *Burnett, W. Emory, 3401 N. Broad St. (40)
1962. Burney, Leroy E., 3400 N. Broad St. (40)
1966. Burns, William P., 3400 Spruce St. (19104)
1954. Burros, Harry M., 419 S. 19th St. (46)
1966. Burstein, Frank, 8541 Bustleton Ave. (19152)
1941. Butler, Miriam, Watersmeet, Glen Mills, Pa.
1957. *Butterworth, Thomas, 411 Walnut St., Reading, Pa.
1965. Byron, Harold J., Wyncote House, Wyncote, Pa.
1966. Cahn, Milton M., 1930 Chestnut St. (19103)
1957. Calhoun, John Alfred, Jr., 214 Elm Ave., Swarthmore, Pa.
1958. Cameron, Charles S., 235 N. 15th St. (2)
1962. Camishion, Rudolph C., 1025 Walnut St. (7)
1952. Campbell, Edward W., Abington Memorial Hospital
1944. Campion, Richard J., Box 136, Springhouse, Ambler, Pa.
1964. Cancelmo, Jesse J., Jr., 337 W. Lancaster Ave., Wayne, Pa.
1967. Carabasi, Ralph A., 255 S. 17th St. (19103)
1935. Carey, Lawrence S., 2241 Garrett Road, Drexel Hill, Pa.
1931. Cariss, Walter L., 202 Avon Rd., Narberth, Pa. (19072)
1930. Carpenter, Chapin, 502 Chestnut Lane, Wayne, Pa.
1959. Carrington, Elsie R., Woman's Medical College (29)
1960. Carroll, Robert T., 1015 Chestnut St., Jefferson Bldg., Room 803 (19107)
1917. Carson, John B., Newtown Square, Pa.
1956. Carty, James Byron, 51 Hampden Rd., Upper Darby, Pa.
1956. Casey, Paul R., 8350 Roosevelt Rd., Bryn Mawr, Pa.
1943. Castigliano, S. Gordon, 853 Mt. Pleasant Rd., Bryn Mawr, Pa.
1949. Caswell, H. Taylor, 3401 N. Broad St. (40)
1954. Catenacci, Alfred J., 343 Winding Way, Merion Station, Pa.
1954. Cathcart, Richard T., Jefferson Hospital, 1025 Walnut St. (7)
1950. *Chamberlain, Richard H., 3400 Spruce St. (4)
1939. Chamberlin, George W., Reading Hospital, Reading, Pa.
1966. Chamblin, William D., 3717 Chestnut St. (19104)
1945. Chance, Burton, Jr., 4400 Baltimore Ave. (19104)
1954. *Charny, Charles W., 2039 Delancey Pl. (3)
1966. Chernoff, Benjamin, 6901 Old York Rd. (19126)
1960. Chirico, Anna-Marie, Hosp. of Univ. of Pa. (4)
1966. Chisum, Melvin J., 245 N. Broad St. (19107)
1947. Chouké, Kehar S., 4050 Forest Hill Ave., Apt. 3, Richmond, Va. (23225)
1945. Cianfrani, Theodore, 1930 Chestnut St. (3)

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1939. Ciccone, Emmet F., 2021 Murray St. (15)
1960. Cinquino, Mario A., 1518 S. Broad St. (46)
1960. Clader, Stanley C., 825-A Glenbrook Ave., Bryn Mawr, Pa.
1951. Clagett, A. Henry, Jr., 515 W. Eighteenth St., Wilmington, Del. (19802)
1966. Clark, Gerald R., Elwyn School, Elwyn, Pa. (19063)
1962. Clark, James E., 1015 Chestnut St. (7)
1951. Clark, John K., 347 Aubrey Rd., Wynnewood, Pa. (19096)
1947. Clark, Thomas Williams, 218 W. Chestnut Hill Ave. (18)
1963. Cleveland, Albert F., 615 Morgan Ave., Drexel Hill, Pa.
1958. Closson, Edward W., 2320 S. Broad St. (45)
1910. Cloud, J. Howard, Alden Park Manor (19144)
1949. Cohen, Louis B., 1320 Arrott St. (24)
1953. Cohen, Robert V., 520 Spring Ave., Elkins Park 17, Pa.
1967. Cohen, Sarle H., 5577 N. Park Ave. (19141)
1965. Cohn, Edwin M., 1351 Tabor Rd. (19141)
1964. Cohn, Herbert E., 1025 Walnut St. (19107)
1934. Collins, Leon Howard, Jr., 133 S. 36th St. (4)
1949. Conger, Kyril B., 3401 N. Broad St. (40)
1961. Coon, Julius M., 1025 Walnut St. (7)
1945. Coonel, Pauline, 47 E. Mt. Pleasant Ave. (19)
1930. *Cooper, David A., 1328 Medford Rd., Wynnewood, Pa. (19096)
1953. *Cooper, Donald R., Woman's Medical College, 3300 Henry Ave. (29)
1960. Cooper, Edward Sawyer, 6710 Lincoln Drive (19)
1947. Coppolino, John F., 1807 S. Broad St. (45)
1952. *Corbit, John D., Jr., 3717 Chestnut St. (4)
1959. Cormeny, George F., 3818 Chestnut St. (4)
1965. Cornelison, Floyd S., Jr., 1025 Walnut St. (19107)
1941. Cornelius, Chalmers E., 119 Summit Ave., Jenkintown, Pa.
1911. Cornell, Walter S., Board of Public Education, Parkway and 21st St. (3)
1914. Corson, Edward F., 3047 Spring Mill Rd., Plymouth Meeting, Pa.
1940. Cowan, Thomas H., 1930 Chestnut St. (3)
1966. Cram, Robert H., 918 County Line Rd., Bryn Mawr, Pa. (19010)
1947. *Crane, A. Reynolds, Pennsylvania Hospital (7)
1937. Crane, Martin P., 4701 Pine St. (43)
1952. Crellin, J. Antrim, 1930 Chestnut St. (3)
1955. Crist, Walter A., 211 N. 5th St., Camden, N. J.
1963. Crosley, Archer P., Jr., The Presbyterian Hospital (4)
1959. Cullen, Milton L., 1936 Cottman Ave. (19111)
1962. Cunningham, John D., 3400 Spruce St. (4)
1934. Custer, R. Philip, 643 Moreno Road, Narberth, Pa.
1958. D'Alonzo, Walter A., 1647 S. 15th St. (45)
1938. Dannenberg, Arthur M., Sr., 235 S. 15th St. (2)
1951. Daugherty, Earl A., 2818 W. Girard Ave. (30)
1953. Davie, John H., 37 S. 20th St. (3)
1947. *Davis, C. Nelson, 524 Howe Rd., Merion Station, Pa.
1936. Davis, David M., 960 County Line Rd., Bryn Mawr, Pa. (19010)
1957. Davis, John Evan, Tedwyn Apts., 840 Montgomery Ave., Bryn Mawr, Pa.
1951. *Davis, J. Wallace, 135 18th St. (3)
1961. Dawber, Bryan A., Penn Mutual Life Insurance Co. (5)
1963. Day, H. James, Temple University Hospital (19140)
1935. *Deaver, J. Montgomery, Lankenau Medical Bldg. (19151)
1957. Decker, John Paul, Pennsylvania Hospital (7)
1957. Dees-Porch, Frances, Glendale Road & Chestnut Sts., Upper Darby, Pa.
1947. Dehne, Theodore L., Adams Avenue and Roosevelt Blvd. (24)
1958. Deitz, George W., III, 2240 Pine Rd., Huntingdon Valley, Pa.
1965. Delaney, William E., III, 11th & Sansom Sts. (19107)

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1955. De Long, Samuel L., 223 Lancaster Ave., Devon, Pa.
1952. DeLuca, Charles Q., 255 S. 17th St. (3)
1952. De Orsay, Ralph H., 1241 Lindale Ave., Drexel Hill, Pa.
1951. *De Palma, Anthony F., 248 S. 21st St. (3)
1963. Derham, Robert J., McClatchy Bldg., Upper Darby, Pa.
1966. deRivas, Carmela F., Stanbridge & Sterigere Sts., Norristown, Pa.
1959. DeTuerk, John J., 1930 Chestnut St. (3)
1959. Dickens, Helen O., P. O. Box 9336 (39)
1958. Dickstein, Benjamin, 6810 Castor (24)
1956. Dietrich, Herbert James, Jr., Hospital of the University of Pennsylvania (4)
1952. Digilio, Victor A., 2200 St. James Place (17)
1963. DiMarino, Anthony J., 735 Delaware St., Paulsboro, N. J.
1960. Dinon, Louis R., 330 S. 9th St. (7)
1920. Doane, Joseph C., 5286 34th Ave., N., St. Petersburg, Fla. (33710)
1948. Dohan, Francis Curtis, 80 Princeton Road, Cynwyd, Pa.
1954. Donaldson, James B., 1901 Walnut St. (3)
1946. Donnelly, Andrew J., 33rd and Powelton Ave. (4)
1937. Donnelly, Robert T. M., 5205 N. Broad St. (41)
1962. Dorman, Philip J., Fidelity Mutual Life Insurance Co. (1)
1964. Dratman, Mary B., Woman's Medical College, Henry Ave. & Abbottsford Road (19129)
1952. Drayer, Calvin S., 111 N. 49th St. (39)
1949. *Dripps, Robert D., 3400 Spruce St. (4)
1944. Dublin, George J., 1930 Chestnut St. (3)
1935. Duncan, Garfield G., 330 S. 9th St. (7)
1966. Duncan, Theodore G., 330 S. Ninth St. (19107)
1953. Dupler, Donald A., 4028 Walnut St. (4)
1966. DuPree, Richard H., 414 Cooper St., Camden, N. J. (08102)
1942. *Durant, Thomas M., Einstein Medical Center, N.D., York & Tabor Rds. (19141)
1942. Dyer, W. Wallace, Bryn Mawr Medical Bldg., Bryn Mawr, Pa.
1962. D'Zmura, Thomas, L., Presidential Apts., City Line Ave. & Presidential Blvd. (19131)
1936. Easby, Mary Hoskins, R. D. 1, Burlington, Vt.
1948. Eaton, Francis H., 4007 Chestnut St. (39)
1964. Eberlein, Walter R., 1740 Bainbridge St. (19146)
1944. Eger, Sherman A., 255 S. 17th St. (3)
1944. Ehrich, William E., 540 W. Hortter St. (19)
1966. Ehrlich, George E., Albert Einstein Medical Center, N.D. (19141)
1956. Eiman, John W., Abington Memorial Hospital, Abington, Pa.
1962. Eisenberg, Samuel W., 3536 N. Broad St. (40)
1959. Eisenhower, James S. D., Jr., 2704 Pacific Ave., Wildwood, N. J.
1958. *Eisman, Sylvan H., 3600 Spruce St. (4)
1959. Elias, Elmer J., 10 Monticello Ave., Trenton, N. J.
1949. Elkinton, J. Russell, Oak Lane, Moylan, Pa.
1961. Elliott, Frank A., 807 Spruce St. (7)
1957. Ellis, Richard A., 255 S. 17th St. (3)
1955. Ellis, Samuel, 275 Bryn Mawr Ave., Bryn Mawr, Pa. (19010)
1939. Ellis, Van Mashburn, 1528 Spruce St. (2)
1928. Ellison, Richard T., Medical Office Bldg., 1245 Highland Ave., Abington, Pa. (19001)
1936. Ellson, J. Vernon, Glendale Road & Chestnut St., Upper Darby, Pa.
1937. *Elsom, Kendall A., Scott Paper Co. (13)
1932. Engel, Gilson C., Lankenau Medical Bldg. (19151)
1945. *English, O. Spurgeon, 449 Righters Mill Rd., Narberth, Pa.
1965. Epstein, Isadore S., 5219 Lebanon Ave. (19131)
1942. Erb, William H., 133 S. 36th St. (4)
1957. Erdman, William James, II, 3400 Spruce St. (4)
1965. Erslev, Allan J., 1015 Sansom St. (19107)
1950. Ersner, Matthew S., 1915 Spruce St. (3)

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* S. Weir Mitchell Associate.

1957. Etzl, Michael M., 9025 Frankford Ave. (14)
1954. Evans, William W., Box 2, Bryn Mawr, Pa.
1955. Eynon, Harold K., Route 70 at East Gate, Barclay Farm, Haddonfield, N. J.
1946. Farell, David M., 1912 Spruce St. (3)
1903. Farr, Clifford B., Railroad Ave. & County Line Rd., Bryn Mawr, Pa.
1945. Farrar, George Elbert, Jr., 528 Scott Rd., Gladwyne, Pa.
1953. Farrell, Harry L., 1930 Chestnut St. (3)
1964. Faust, Herbert A., 245 N. Broad St. (19107)
1935. *Ferguson, Lewis Kraeer, 419 S. 19th St. (46)
1936. *Fetter, Ferdinand, 322 S. 21st St. (3)
1945. Fetter, John S., 1980 Country Club Dr., Huntingdon Valley, Pa. (19006)
1952. *Fields, Harry, 133 S. 36th St. (4)
1959. Fineberg, Charles, 255 S. 17th St. (3)
1955. Finestone, Albert J., 3701 North Broad St. (40)
1960. Finkbiner, Rodman B., 811 Spruce St. (7)
1943. *Finkelstein, Arthur K., Graduate Hospital (46)
1955. Finkelstein, David, 419 S. 19th St. (46)
1955. *Finley, John K., 527 Welsh St., Chester, Pa.
1954. *Finn, Joseph L., 8014 Burholme Ave. (11)
1947. First, Arthur, 1714 Spruce St. (3)
1954. Fischer, Carl C., Girard College (21)
1950. *Fischer, H. Keith, School Lane House, 5450 Wissahickon Ave. (19144)
1962. Fisher, George R., III, 801 Spruce St. (7)
1958. Fisher, Robert M., 118 Airdale Rd., Rosemont, Pa.
1956. Fite, Franklin K., Germantown Hospital (44)
1952. Fitts, William T., Jr., 3400 Spruce St. (4)
1952. Flexner, Louis B., 248 Anatomy-Chemistry Bldg., Univ. of Pa. (4)
1928. *Flick, John B., 819 Black Rock Rd., Gladwyne, Pa.
1939. Flippin, Harrison F., Lankenau Hospital (51)
1957. Fornwalt, George R., 47 Copley Rd., Upper Darby, Pa.
1954. Forster, H. Walter, Jr., 37 S. 20th St. (3)
1960. Foulger, John H., 601 Rockwood Rd., Wilmington, Del. (2)
1941. Fox, C. Calvin, 1806 Spruce St. (3)
1963. Fox, Eva F., Woman's Medical College (19129)
1963. Frank, Paul E., York Road & Summit Ave., Hatboro, Pa.
1963. Frank, Robert L., 1282 Rydal Rd., Rydal, Pa. (19046)
1955. Frayer, William C., 37 S. 20th St. (3)
1941. Frazier, William Doane, 419 Gilpin Rd., Narberth Pa.
1952. Freed, Herbert, 255 S. 17th St. (3)
1956. Freeman, Joseph T., 8-A Rittenhouse Plaza (3)
1963. Freiman, Henry D., 1500 Hellerman St. (49)
1958. Freiwald, Milton J., 222 S. 19th St. (3)
1964. French, Gordon N., Misericordia Hospital (19143)
1955. Friedenberg, Zachary B., 133 S. 36th St. (4)
1951. Friedman, Paul Sigmund, 1422 Chestnut St. (2)
1960. Friedman, Sidney, 1740 Bainbridge St. (46)
1958. Frisch, Frederick, 888 Glenbrook Ave., Bryn Mawr, Pa.
1937. Fritz, Herbert H., Pennswood Rd., Bryn Mawr, Pa.
1961. Frobese, Alfred S., Abington Memorial Hosp., Abington, Pa.
1953. Fruchter, Joseph M., The Philadelphian, 2401 Pennsylvania Ave. (19130)
1940. Fry, Kenneth E., 1719 Rittenhouse Sq. (3)
1935. Fry, Wilfred Eyles, 1930 Chestnut St. (3)
1963. Furgiuele, Francis P., 5430 Greene St. (19144)
1937. Furlong, Thomas F., Jr., Times Medical Building, Ardmore, Pa.
1964. Galligan, William J., 2909 Garrett Road, Drexel Hill, Pa.
1954. Gambescia, Joseph M., 1422 Race St. (2)

* S. Weir Mitchell Associate.

1966. Garcia, Celso-Ramon, University Hospital (19104)
1951. Garlichs, Richard W., 216 N. Manoa Road, Havertown, Pa.
1931. Garner, Vaughn C., 477 East Wadsworth St. (19)
1953. Garnet, James D., 807 Spruce St. (7)
1959. Gartland, John J., 269 S. 19th St. (3)
1950. Garvin, Eugene J., 533 S. 46th St. (43)
1952. Gefter, William I., 1900 Rittenhouse Square (3)
1935. Geist, Donald C., 301 E. City Line, Bala-Cynwyd. Pa.
1955. Gelfand, David, 1722 Pine St. (3)
1965. Georges, Thomas W., Jr., The Health & Welfare Bldg., 7th & Forster Sts., Harrisburg, Pa. (17102)
1941. *Gershon-Cohen, J., 255 S. 17th St. (3)
1959. Gerson, Irvin M., Cedarbrook Apts., Chearbrook Hills, Wyncote, Pa. (19095)
1955. Gettes, Bernard C., 128 Overhill Rd., Cynwyd, Pa.
1963. Giambalvo, Giacchino P., 818 N. 63rd St. (19151)
1965. Gianakon, Harry G., 111 N. 49th St. (19139)
1934. *Gibbon, John H., Jr., 1025 Walnut St. (7)
1939. *Gibson, Glen Gregory, 2031 Locust St. (3)
1944. Gifford, Edward S., Jr., 1913 Spruce St. (3)
1956. Gilbert, Philip D., Cooper Hospital, Camden, N. J.
1947. Giletto, Basil J., 2020 Locust St. (3)
1955. Gill, Robert J., 801 Spruce St. (7)
1922. Gilmour, William R., 6616 Woodland Ave. (42)
1952. Gilpin, Sherman F., Jr., 1023 Beury Building, 3701 N. Broad St. (40)
1963. Gindhart, Floyd D., 1213 Hamilton Avenue, Trenton, N. J.
1965. Ginley, Thomas H., Jr., The Iroquois, 47th St. & City Line Ave. (19151)
1949. Ginsburg, Isadore W., 3401 N. Broad St. (40)
1958. Girsh, Leonard S., 3701 N. Broad St. (40)
1954. Gislason, Gerhard J., 245 N. Broad St. (7)
1964. Gloeckner, M. Louise C., 110 E. Fourth Ave., Conshohocken, Pa.
1956. Goldberg, Harry, 7913 Rolling Green Rd., Cheltenham, Pa.
1965. Goldburgh, Warren P., 1932 Spruce St. (19103)
1958. Golub, Leib J., 136 S. 16th St. (2)
1963. Goluboff, Bernard, 413 W. Mermaid Lane (19118)
1965. Goodman, David H., 6816 Castor Ave. (19149)
1956. Goracci, Armando F., 133 S. 36th St. (19104)
1937. Gordon, William, 5345 Spruce St. (39)
1964. Gottlieb, Harry, 5603 Greene St. (19144)
1964. Gottlieb, Philip M., 818 Medical Arts Bldg., 16th & Walnut Sts. (19102)
1965. Gould, R. John, Delaware County Memorial Hospital, Drexel Hill, Pa.
1956. *Gouley, Benjamin, 5577 N. Park Ave. (41)
1966. Gowen, George F., Misericordia Hospital (19143)
1957. Grahn, Henry Victor, 5132 Chester Ave. (43)
1966. Grant, B. David, 918 County Line Road, Bryn Mawr, Pa. (19010)
1926. Grant, Francis Clark, 750 Germantown Pike, Lafayette Hill, Pa.
1965. Grassi, Michael O. A., 2038 Locust St. (19103)
1965. Greenbaum, Charles H., 6810 Castor Ave. (19149)
1940. Greene, Lloyd B., 803 Spruce St. (7)
1952. *Greening, Roy R., Dept. of Radiology, Jefferson Hospital (7)
1958. *Greenspan, Benjamin, 1319 W. Tabor Rd. (41)
1955. Greenstein, Raphael H., 1710 Pine St. (3)
1941. *Groff, Robert A., Univ. of Pa. Hospital (4)
1952. *Grotzinger, Paul J., 2121 Valley Rd., Huntingdon Valley, Pa.
1952. *Grove, D. Dwight, 5025 N. Marvine St. (19141)
1966. Guthrie, Marshall B., 1500 Spring Garden St. (19101)
1945. György, Paul, Philadelphia General Hospital (19104)

* S. Weir Mitchell Associate.

1935. Hadden, Samuel B., 135 S. 19th St. (19103)
1945. Hahn, George A., 255 S. 17th St. (3)
1964. Hall, John H., 3401 N. Broad St. (19140)
1955. Hallahan, John D., 104 W. Front St. Media, Pa.
1951. Hallett, Joseph W., 136 S. 16th St. (2)
1961. Hammett, Van Buren O., 249 N. Broad St. (19107)
1954. Hand, B. Marvin, 1801 Pennsylvania Blvd. (3)
1938. Hand, John G., 1724 Pine St. (3)
1964. Hanes, Robert B., 501 Long Lane, Upper Darby, Pa.
1947. Hanes, William James, 218 E. Beechtree Lane, Wayne, Pa.
1963. Hanhausen, Edward H., 418 E. Lancaster Ave., Wayne, Pa.
1950. Hansen, A. Victor, 910 Centennial Rd., Penn Valley, Narberth, Pa. (19072)
1957. Harasym, Emil L., 5416 North 5th St. (20)
1956. Harbert, Frederick, Jefferson Hospital (7)
1952. Harper, W. Benson, State Rd. & Rogers Ave., Bywood, Pa.
1951. Harkins, Herbert P., 1930 Chestnut St. (3)
1966. Harner, Richard N., Graduate Hospital (19146)
1966. Harrington, Edwin D., Jefferson Medical College (19107)
1963. Harris, Charles, 5301 Old York Road (41)
1962. Harris, James R., 3222 W. Penn St. (19129)
1959. Harris, James S. C., 5401 Wayne Ave. (44)
1948. Harris, T. N., 5112 Woodbine Ave. (31)
1929. Harrison, Francis G., 1900 Spruce St. (3)
1964. Hart, Alonzo W., 847 N. 63rd St. (19151)
1926. Hartmann, Fred I., 1914 Pine St. (3)
1947. Harvey, Rolfe Marsh, 669 Mill Rd., Villanova, Pa.
1961. Harvie, Fred H., 3400 Spruce St. (4)
1935. *Haskell, Benjamin F., 1427 Spruce St. (2)
1958. Haupt, George J., Lankenau Medical Bldg. (51)
1960. Hausman, David H., Pennsylvania Hospital, 8th & Spruce Sts. (7)
1947. Havens, Walter Paul, Jr., 139 Cheuold Lane, Haverford, Pa.
1963. Hayes, Merrill B., 710 Madison St., Chester, Pa.
1952. Hayllar, Benjamin L., 803 Spruce St. (7)
1961. Hayward, Malcolm L., 111 N. 49th St. (39)
1961. Heath, Frederick K., Merck Sharp and Dohme, West Point, Pa.
1964. Heath, Robert D., Lankenau Medical Bldg. (19151)
1957. Hedges, Thomas R., Jr., 330 South 9th St. (7)
1961. Heggestad, Gilman E., Conestoga Medical Bldg., Bryn Mawr, Pa.
1965. Heil, Charles G., Jr., The Benson East, Jenkintown, Pa. (19046)
1965. Heine, William I., 5579 N. Park Ave. (19141)
1944. Herbut, Peter A., Dept. of Pathology, Jefferson Medical College Hospital (7)
1954. Herron, James R., 1486 Haddon Ave., Camden 3, N. J.
1963. Hesch, Joseph A., Misericordia Hospital (19143)
1949. Heyl, W. Meredith, 100 W. Coulter St. (44)
1961. Hilferty, Daniel J., Jr., Sutton Terrace Apts., 50 Belmont Ave., Bala-Cynwyd, Pa.
1952. Hinkson, DeHaven, 329 N. 40th St. (4)
1962. Hinman, E. Harold, 1025 Walnut St. (7)
1924. Hirst, John C., 2d., 2016 Rittenhouse Sq. (19103)
1943. Hitschler, William J., Bethlehem Pike & Summit (18)
1944. Hneleski, Ignatius S., 802 S. 48th St. (43)
1943. Hockaday, Agnes, 316 Shadeland Ave., Drexel Hill, Pa.
1939. *Hodes, Philip J., Dept. of Radiology, Jefferson Medical College (7)
1947. *Hodges, John H., 1025 Walnut St. (7)
1963. Hoffman, Carl J., 2901 Cottman Ave. (19149)
1959. Hoffman, Francis H., 3401 N. Broad St. (40)
1948. Hoffman, George L., Jr., 133 S. 36th St. (4)

* S. Weir Mitchell Associate.

1965. Holfelner, Edward D., Chestnut St. & Ashby Road, Upper Darby, Pa.
1954. Hollander, George, 3500 Vista St. (36)
1949. Hollander, Joseph L., 36th & Spruce Sts. (4)
1964. Holling, Herbert E., 3400 Spruce St. (19104)
1951. Hollis, Charles B., 28 W. School Lane (44)
1952. Holloway, Edward E., 1736 Pine St. (3)
1966. Holly, Roy G., 1025 Walnut St. (19107)
1966. Holter, F. Robert, 1911 Spruce St. (19103)
1949. Hopkins, Henry U., Garden Court Plaza, 47th & Pine Sts. (43)
1953. Horan, Charles A., 734 N. 63rd St. (19151)
1946. Horan, Gerald W., Marple-Newtown Medical Bldg., Newtown Square, Pa.
1947. *Horwitz, Orville, Hospital of the University of Pennsylvania (4)
1950. Houlihan, Carl T., 207 McClatchy Bldg., Upper Darby, Pa.
1944. Housel, Edmund L., 255 S. 17th St. (3)
1958. *Howard, John M., 230 N. Broad St. (2)
1964. Howland, Alvin Wilcox, School Lane House (44)
1946. Howson, John Yeatts, 316 Lancaster Ave., Wayne, Pa.
1951. Hubbard, John P., 133 S. 36th St. (19104)
1947. *Huber, John Franklin, Temple University School of Medicine (40)
1937. Hughes, Joseph, 111 N. 49th St. (39)
1939. Hughes, P. Boland, 419 Hillbrook Rd., Bryn Mawr, Pa.
1951. Hundley, J. Warren, 3818 Chestnut St. (4)
1954. Hunsicker, William C., Jr., 255 S. 17th St. (3)
1946. Hunt, William T., Jr., 6 Penn Center Plaza (3)
1921. Hunter, Robert John, 928 N. 63rd St. (19151)
1952. Hyman, Harold L., 3401 N. Broad St. (40)
1950. *Imbriglia, Joseph E., 934 Cedar Grove Rd., Wynnewood, Pa. (19096)
1949. Ingraham, Norman R., Jr., Stenton and Whitemarsh Aves. (18)
1957. Isaacson, Howard, Haverford Ave. & Drexel Rd. (19151)
1951. *Isard, Harold J., Albert Einstein Medical Center, York & Tabor Rds. (19141)
1951. *Israel, Harold L., 302 S. 19th St. (19103)
1947. Israel, S. Leon, 807 Spruce St. (7)
1947. Jaeger, Rudolph, Jefferson Bldg., 1015 Chestnut St. (7)
1966. Jannett, Andrew F., Packard St., Hammonton, N. J.
1965. Jenkins, B. Wheeler, 1526 E. Upsal St. (19150)
1960. Jenson, William K., Pennsylvania Hospital (19107)
1943. *Johnson, Julian, 3400 Spruce St. (4)
1959. Johnson, Robert G., 1015 Walnut St. (7)
1938. Johnson, Thomas A., 135 S. 18th St. (3)
1952. Jonas, Karl C., 916 19th St., N.W., Washington, D.C. (20006)
1963. Jones, Robert E., 111 N. 49th (39)
1963. Joseph, Rosaline R., 3401 N. Broad St. (40)
1966. Joyce, John J., III, 5908 Greene St. (19144)
1961. Joyner, Claude R., Jr., 3400 Spruce St. (4)
1957. Kacher, Leon, 2037 Pine St. (3)
1957. Kane, Sydney H., 3458 Cottman Ave. (49)
1948. Kaplan, Louis, 2040 Pine St. (3)
1964. *Kaplan, S. Richard, 419 S. 19th St. (19146)
1938. Kasper, Kelvin A., 135 S. 18th St. (3)
1966. Kassay, Dezso, 1 Summit St. (19118)
1958. Kasser, Max D., 101 S. 20th St. (19103)
1945. Katz, G. Henry, 111 N. 49th St. (39)
1941. Kauffman, M. Luther, Medical Arts Bldg., Jenkintown, Pa.
1947. Kay, Calvin Frederick, Univ. of Pa. Hospital (4)
1954. Kaye, Robert, Children's Hospital, 1740 Bainbridge St. (46)
1947. Keefer, George Pfahler, 136 S. 16th St. (2)

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1963. Keeley, Francis X., Professional Bldg., 1533 Haddon Ave., Camden, N. J.
1966. Keeney, Arthur H., Wills Eye Hospital (19130)
1949. Keiserman, Joseph, 1900 John F. Kennedy Blvd. (19103)
1959. Keitel, Hans G., Ortho Research Foundation, Raritan, N. J. (08869)
1965. Kellow, William F. T., 235 N. 15th St. (19102)
1932. *Kelly, Herbert T., 1830 Delancey Place (19103)
1961. Kelly, William E., 111 N. 49th St. (39)
1965. Kennedy, Charles, 1740 Bainbridge St. (19146)
1941. Kennedy, Patrick J., 32 Hampden Rd., Upper Darby, Pa.
1948. Kern, Franklin Moore, 903 Nicholson Rd., Wynnewood, Pa.
1921. *Kern, Richard A., Temple University Hospital, 3401 N. Broad St. (40)
1935. Keyes, Baldwin L., 2031 Locust St. (3)
1936. King, Orville C., 330 S. 9th St. (7)
1952. Kirshner, Jacob J., 1930 Chestnut St. (3)
1960. Kitchell, J. Roderick, Abington Hospital Medical Office Bldg., 1245 Highland Ave., Abington, Pa. (19001)
1920. Klein, Thomas, 250 S. 18th St. (3)
1952. Kleinbart, Morris, 1922 Pine St. (3)
1954. Kligman, Albert M., 36th & Spruce Sts. (4)
1934. Kline, Oram R., 514 Cooper St., Camden, N. J.
1959. Kline, Oram R., Jr., 406 Cooper St., Camden 2, N. J.
1948. Klingensmith, Paul O., Med. Science Bldg., Lankenau Hosp. (19151)
1958. Klingensmith, Walter C., 3600 Spruce St. (4)
1956. Klinghoffer, June F., Woman's Medical College (29)
1960. Knopf, Carl L., 5312 Spruce St. (39)
1960. Knorr, John K., 3rd Lankenau Medical Bldg. (19151)
1959. Knowles, Harry J., 255 S. 17th St. (3)
1945. Kobler, H. B., School Lane House, 5450 Wissahickon Ave. (19144)
1959. Koiwai, Eichi Karl, 235 N. 15th St. (2)
1963. Kolbe, Henry W., Philadelphia General Hospital (19104)
1957. Koltes, John A., 530 Spring Lane (19128)
1936. Konzelmann, Frank Williamson, Shore Memorial Hosp., Somers' Point, N. J.
1949. *Koop, C. Everett, 1740 Bainbridge St. (46)
1964. Koppel, Max M., 7310 Castor Ave. (19115)
1952. Kornblueh, Igho H., Welsh Rd. & Verree Rd. (15)
1958. Kovach, Coleman W., 2031 Locust St. (3)
1941. Krall, J. Thomas, 85 N. Lansdowne Ave., Lansdowne, Pa.
1948. Kramer, David W., 2007 Pine St. (3)
1954. *Krasnoff, Sidney O., Tabor Medical Bldg., York & Tabor Roads (41)
1952. Kravitz, Charles H., 626 Ashbourne Rd., Elkins Park, Pa.
1964. Kremens, Victor, 60 E. Township Line Road, Elkins Park, Pa.
1949. Kressler, Robert J., 330 S. 9th St. (7)
1941. Krewson, William E., 3rd, 1930 Chestnut St. (3)
1947. Kuhlenbeck, Hartwig, Woman's Medical College, 3300 Henry Ave. (29)
1964. Kustrup, John F., 1418 S. Broad St., Trenton, N. J.
1947. La Boccetta, Alfred Charles, Philadelphia General Hospital, 34th St. & Curie Ave. (4)
1952. *Lachman, John W., 3401 N. Broad St. (40)
1963. LaFia, David J., 550 N.E. 56th St., Miami, Fla. (33137)
1951. Lame, Edwin L., 518 Auburn Ave. (19118)
1950. Lamont, Auston, 3400 Spruce St. (4)
1946. Lampe, William T., 4207 Tyson Ave. (35)
1962. Landis, Harry P., Jr., 901 Columbia Ave., Palmyra, N. J.
1963. Lang, Leonard P., 1009 Park Place, Wilmington, Del.
1952. *Lang, Warren R., 1025 Walnut St. (19107)
1966. Langfitt, Thomas W., Pennsylvania Hospital (19107)
1941. Langner, Paul H., Jr., 1208 Edmonds Ave., Drexel Hill, Pa.

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* S. Weir Mitchell Associate.

1945. Lansbury, John, 3401 N. Broad St. (40)
1926. Laws, George M., 1907 Spruce St. (3)
1967. Lear, Walter J., Phila. Dept. of Public Health, 540 Municipal Services Bldg. (19107)
1950. Learner, Norman, 3701 N. Broad St. (40)
1920. Leavitt, Frederic Headley, 1527 Pine St. (2)
1941. Leberman, Paul R., Hospital of the University of Pennsylvania (4)
1957. Lee, Charles Trumbull, Jr., 33 E. Chestnut Hill Ave. (18)
1952. *Lehman, J. Stauffer, 230 N. Broad St. (2)
1961. Leibfried, Jane M., 5501 Green St. (44)
1951. Leivy, Frank. E., Wm. Penn House, 1919 Chestnut St. (19103)
1944. Lell, William A., 333 S. 18th St. (3)
1935. Lemmon, William T., 133 S. 36th St. (4)
1965. Leopold, Howard C., 4623 York Rd. (19140)
1955. Leopold, Robert L., 269 S. 19th St. (3)
1967. Leslie, W. Munroe, 8800 Germantown Ave. (19118)
1944. Lettiere, Anthony J., 425 E. State St., Trenton, N. J.
1965. Levenson, Carl, Albert Einstein Medical Center, N. D. (19141)
1933. Levering, J. Walter, Beaver Hill Apts., Jenkintown, Pa. (19046)
1959. Levick, Leonard J., 1300 E. Cliveden St. (19)
1958. Levine, Samuel, 7100 N. 19th St. (26)
1964. Levit, Edithe J., 3930 Chestnut St. (19104)
1964. Levit, Samuel M., 1910 Spruce St. (19103)
1958. LeWinn, Edward B., Upper Black Eddy, Pa. (18972)
1915. Lewis, Fielding O., Providence Road, Media, Pa.
1960. *Lewis, George C., Jr., 230 N. Broad St. (2)
1967. Lewis, Stuart H., 2050 W. Chester Ave., Havertown, Pa. (19083)
1964. Leymaster, Glen R., Woman's Medical College, 3300 Henry Ave. (19129)
1955. Lieberman, George E., 269 S. 19th St. (19103)
1956. Likoff, William, 249 N. Broad St. (2)
1962. Lilley, George W., Scott Paper Co. (19113)
1940. Limberger, William A., 301 S. Church St., West Chester, Pa.
1958. Lin, David Y. P., 2222 S. Broad St. (45)
1966. Lin, Paul M., The Benson, Suite 123, Jenkintown, Pa. (19046)
1938. Lndauer, M. August, 133 S. 36th St. (4)
1956. Linquist, John N., 422 Sabine Ave., Wynnewood, Pa.
1927. Lindsey, Walter H., 102 Park Drive, Highland Park, Doylestown, Pa.
1936. Lintgen, Charles, 1930 Chestnut St. (3)
1958. Lipshutz, Arthur, 7974 Fayette St. (19)
1959. *Lipshutz, Herbert, 807 Spruce St. (19107)
1953. Lipsius, Eward I., 319 S. 16th St. (3)
1959. Little, Ralph B., 111 N. 49th. (39)
1944. Livesey, Mary M., 8835 Germantown Ave. (19118)
1936. Llewellyn, Thomas H., 910 N. 63rd St. (19151)
1907. Lodholz, Edward, The Fairfax, 43rd & Locust Sts. (4)
1963. London, Russell I., 6800 Castor Ave. (49)
1933. Long, Esmond R., Society Hill Towers, 23B, 220 Locust St. (19106)
1951. Long, Joseph P., 1930 Chestnut St. (3)
1935. Long, William L., 2038 Locust St. (3)
1931. Longaker, Edwin P., 41 E. Montgomery Ave., Ardmore, Pa.
1951. Lorber, Stanley H., Temple Univ. Hospital, 3401 N. Broad St. (19140)
1961. Lorry, Ralph W., 1247 E. Luzerne St. (24)
1960. Lovett, B. Frank, North Park Drive & Airport Highway, Pennsauken, N. J. (08109)
1963. Lubowitz, Richard M., Benson East Apts., Jenkintown, Pa.
1936. Lucchesi, Pascal Francis, 601 E. Gorgas Lane (19)
1960. Lucine, Albert A., 345 S. 19th St. (3)
1939. Luongo, Romeo A., 2054 Locust St. (3)
1951. Luscombe, Herbert A., 136 S. 16th St. (2)

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1952. Lutman, Frank C., 100 W. Coulter St. (44)
1919. Lynch, Frank B., Jr., 809 Cambridge Apts., School House Lane (44)
1950. Lynch, J. Edward, Suite 305, Wynnewood House, 300 E. Lancaster Ave., Wynnewood, Pa.
1953. Lyons, John W., 255 S. 17th St. (3)
1965. McCabe, James L., Jr., Barclay Bldg., One Belmont Ave., Bala-Cynwyd, Pa.
1932. *McCahey, James F., Rittenhouse-Claridge, 18th & Walnut Sts. (3)
1965. McCarty, Daniel J., Jr., 230 N. Broad St. (19102)
1958. McClenahan, John L., 39 East Chestnut Hill Ave. (18)
1935. McClenahan, William U., 33 East Chestnut Hill Ave., Chestnut Hill (18)
1957. McConnell, Edward L., Jr., 2342 S. Broad St. (45)
1934. McCouch, Grayson P., Univ. of Pa. School of Medicine (4)
1962. McCracken, Stewart, 3701 N. Broad St. (40)
1938. McCrea, Lowrain E., 1930 Chestnut St. (3)
1958. McCune, Wallace G., 5555 Wissahickon Ave. (44)
1942. McDonald, P. Robb, Lankenau Medical Bldg. (19151)
1953. McDonnell, William V., West Jersey Hospital, Camden 4, N. J.
1952. McElroy, Robert C., 133 S. 36th St. (4)
1950. McFadden, William M., 1187 E. Washington Lane (38)
1949. McGavic, John S., 601 Montgomery Ave., Bryn Mawr, Pa.
1945. McGee, Lemuel Clyde, Hercules Powder Company, Wilmington, Del.
1954. McGehee, Edward H., 33 E. Chestnut Hill Ave. (18)
1952. McGlade, Thomas H., 719 Cooper St., Camden, N. J.
1966. McGowan, Larry, Fitzgerald Mercy Hospital, Darby, Pa. (19023)
1956. McGrath, Raymond J., 1720 Spruce St. (3)
1959. McKeown, John J., Jr., 255 S. 17th St. (19103)
1933. McLaughlin, James S., 330 S. 9th St. (19107)
1966. McMichael, Harrison, 203 Medical Bldg., Univ. of Pa. (19104)
1924. McMillan, Thomas M., 1901 Walnut St. (3)
1924. McPhedran, F. Maurice, Cope House, Awbury Arboretum (19138)
1955. McStravog, Lawrence J., 6910 Market St., Upper Darby, Pa.
1954. MacFadyen, Bruce V., 1819 Pennsylvania Blvd. (3)
1932. *Macfarlane, Catherine, 136 S. 16th St. (2)
1964. Macht, Elmer L., Jr., York & Keith Roads, Abington, Pa.
1949. MacNeal, Perry Scott, Suite 308, Franklin Medical Bldg., 829 Spruce St. (19107)
1951. MacNeish, W. J., 821 Great Springs Rd., Rosemont, Pa. (19010)
1955. Macy, Dorothy, 705 Beechwood Road, Media, Pa.
1957. Madow, Leo, Woman's Medical College, 3300 Henry Ave. (19129)
1935. Maeder, LeRoy M. A., 1910 Rittenhouse Sq. (3)
1967. Magee, Joseph H., Jefferson Medical College (19107)
1952. Mahoney, J. Francis, 250 S. 18th St. (3)
1965. Mancall, Elliott L., 230 N. Broad St. (19102)
1959. Mandel, Martin M., Benson Manor, Township Line & Washington Lane, Jenkintown, Pa.
1966. Manges, W. Bosley, 1719 Rittenhouse Sq. (19103)
1954. Manges, Willis E., Methodist Hospital, Broad & Wolf Sts. (48)
1949. Manlove, Francis R., 500 Williamson Rd., Gladwyne, Pa.
1960. Manning, Valentine R., 3336 Aldine St. (36)
1956. Manstein, George, 1351 Tabor Rd. (41)
1961. Mapow, Abraham B., 535 Pine St. (6)
1953. Marbach, A. Herbert, 1307 Tabor Rd. (41)
1948. Marden, Philip Ayer, University Hospital, 36th & Spruce Sts. (4)
1958. Marino, Daniel J., 422 East 22nd St., Chester, Pa.
1966. Markind, Simon, Pennsylvania Hospital (19107)
1950. Marshall, E. Wayne, 907 Pine St. (19107)
1951. *Martin, William L., Hahnemann Hospital (2)
1953. Marx, Halvey E., 76 Brennan Drive, Bryn Mawr, Pa.

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1966. Mastroianni, Luigi, Jr., University Hospital (19104)
1939. May, Hans, 255 S. 17th St. (3)
1951. Mayock, Robert L., 3600 Spruce St. (4)
1958. Mays, Ralph Whiteman, 154 W. Tulpehocken St. (44)
1961. Mechanick, Philip, 215 Clwyd Rd., Bala-Cynwyd, Pa. (19004)
1952. Mecray, Paul, Jr., 405 Cooper St., Camden, N. J. (08102)
1951. Medinger, Frederick G., 1941 Woodland Road, Abington, Pa.
1959. Medoff, Joseph, 5400 Wynnefield Ave. (31)
1941. Mendell, Theodore H., 2023 Spruce St. (3)
1943. Meranze, David R., 1031 Lakeside Ave., (26)
1954. Mertens–Roesler, Elizabeth, 2910 Woodpipe Lane (29)
1956. Merves, Louis, 1015 Chestnut St. (7)
1955. Mervine, Thomas B., 1120 Wynnewood Rd., Haddonfield, N. J. (08033)
1959. Metzger, Harry N., 1815 Pine St. (3)
1964. Meyer, Edward C., Fitzgerald Mercy Hospital, Darby, Pa. (19023)
1955. Meyer, Eugene A., 1438 Mt. Ephraim Ave., Camden, N. J.
1937. Meyer, George Phillip, 108 4th Ave., Haddon Hts., N. J.
1964. Michaile, Kenneth I., 1930 Chestnut St. (19103)
1956. Miller, Bernard Joseph, 6013 Greene St. (44)
1935. Miller, Ford A., 4016 Chestnut St. (4)
1939. Miller, Malcolm W., Lankenau Medical Bldg. (19151)
1939. *Miller, Merle M., 6013 Greene St. (44)
1921. Miller, T. Grier, 133 S. 36th St. (4)
1955. Miller, William H., Fitzgerald Mercy Hospital, Darby, Pa.
1965. Mills, Lewis C., Hahnemann Medical College & Hospital, 230 N. Broad St. (19102)
1952. Minehart, John R., 615 E. Allegheny Ave. (19134)
1952. *Mintz, S. S., 1930 Chestnut St. (3)
1963. Mishkin, Mark M., Univ. of Pa. Hospital (19104)
1950. Mitchell, John McK., 6 Bryn Mawr Ave., Bryn Mawr, Pa. (19010)
1952. Mitchell, Robert McNair, 807 Spruce St. (7)
1959. Mitterling, Robert C., Chestnut St. & Glendale Rd., Upper Darby, Pa.
1936. Montgomery, Hugh, 119 Glenn Road, Ardmore, Pa.
1936. *Montgomery, John B., 1930 Chestnut St. (3)
1932. Montgomery, Thaddeus L., 2031 Locust St. (3)
1935. *Moore, John Royal, 3401 N. Broad St. (40)
1947. Moore, Matthew Thibaud, 1813 Delancey Pl. (3)
1958. Moore, Samuel R., Jr., 1600 Arch St. (1)
1958. Morani, Alma D., 3665 Midvale Ave. (29)
1936. Morgan, David R., 218 W. Mt. Carmel Ave., Glenside, Pa.
1944. Morris, Harold H., St. Clair Drive, St. Simons Island, Georgia (31522)
1945. Morris, I. Paul, 1939 Cheltenham Ave. (17)
1965. Morse, Dryden P., 245 N. Broad St. (19107)
1964. Most, William, 994 Haddon Ave., Collingswood 7, N. J.
1957. Moyer, David G., 701 W. Main St., Lansdale, Pa.
1959. Moyer, John H., 230 N. Broad St. (2)
1941. *Mucklé, Craig Wright, 8016 Seminole Ave. (19118)
1929. Mudd, Stuart, Department of Bacteriology, Univ. of Pa. School of Medicine (4)
1960. Mulberger, Robert D., 1930 Chestnut St. (3)
1965. Muller, Otto, Fitzgerald-Mercy Hospital, Darby, Pa.
1930. Murphy, Douglas P., 601 S. Bowman Ave., Merion Station, Pa.
1953. Murphy, Edward J., Bryn Mawr Medical Bldg., Bryn Mawr, Pa.
1958. Murphy, John J., 3600 Spruce St. (4)
1959. Murray, Edwin N., 130 N. Broadway, Camden, N. J.
1955. Murtagh, Frederick, Jr., 3401 N. Broad St. (40)
1951. Myers, David, 3701 N. Broad St. (40)
1965. Myers, Richard N., 307 Lankenau Medical Bldg. (19151)
1955. Nachod, Grace R., 5501 Greene St. (44)

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1945. Naide, Meyer, 2034 Spruce St. (3)
1951. Naidoff, David, 3500 Tudor St. (36)
1966. Nance, Maurice R., 1500 Spring Garden St. (19101)
1950. Nardone, Anthony A., 912 S. 49th St. (43)
1959. Neal, Hunter, S., 406 Lankenau Medical Bldg. (19151)
1958. Nealon, Thomas, F., Jr., 1025 Walnut St. (7)
1948. Neese, Paul H., 17 Derwen Road, Cynwyd, Pa.
1963. Nelson, Arthur D., 3400 N. Broad St. (40)
1943. Nelson, Waldo E., 615 Moreno Rd., Narberth, Pa.
1955. *Nemir, Paul, Jr., 19th & Lombard Sts. (19146)
1935. *Nicholson, Jesse Thompson, 1740 Bainbridge St. (46)
1960. Nicholson, John W., III, 115 Chestnut St., Moorestown, N. J.
1947. Noone, Ernest L., R. D. #1, Elverson, Chester Co., Pa. (19520)
1954. Norris, Charles M., 3401 N. Broad St. (40)
1938. *Norris, Robert F., 513 Wynnewood Ave., Wynnewood, Pa.
1957. Oakey, Richard S., Jr., 309 Medical Bldg., Lankenau Hospital (51)
1963. O'Hara, A. Edward, Jefferson Medical College Hospital (19107)
1947. O'Keefe, John J., 255 South 17th St. (3)
1954. Olsen, Axel K., 230 N. Broad St. (2)
1949. O'Neill, James F., 1425 Woodland Rd., Rydal, Pa. (19046)
1954. O'Neill, Thomas J. E., Centennial Bldg., Front St. & Lehigh Ave. (25)
1947. Ornsteen, A. M., 2007 Delancey Pl. (3)
1955. Ottenberg, Donald J., Beury Bldg., 3701 N. Broad St. (40)
1961. Owen, Barbara J., Bryn Mawr Hospital, Bryn Mawr, Pa.
1949. *Padis, Nicholas, Lankenau Medical Bldg. (51)
1909. Parish, Benjamin Dores, Cresheim Arms Hotel, Allens Lane & Bryan St. (19)
1963. Parish, Benjamin D., Jr., 1201 Bethlehem Pike, Flourtown, Pa.
1945. Park, Felix Roman, 1980 Utica Sq., Tulsa, Okla.
1940. Parker, Alan P., Bryn Mawr Medical Bldg., Bryn Mawr, Pa.
1948. Parkhurst, Leonard Woods, 330 S. 9th St. (7)
1952. Paxson, Newlin F., 245 N. Broad St. (7)
1932. Payne, Franklin L., Univ. of Pa. Hospital (4)
1965. Pearlstine, Beatrice, 7404 Mountain Ave. (19126)
1954. Pearce, Alexander E., 1420 Race St. (2)
1958. Pearce, Rowan C., Jr., 327 Cooper St., Camden 2, N. J.
1943. Pearson, Gerald H. J., Presidential Apts.—Adams House (31)
1954. Pearson, Manuel M., 111 N. 49th St. (39)
1952. Peck, Roy I., 3400 Spruce St. (4)
1960. Peltz, William L., 111 N. 49th St. (39)
1930. *Pendergrass, Eugene P., Radiology Dept., University Hospital, 3400 Spruce St. (19104)
1953. Penneys, Raymond, University of Pennsylvania Hospital (4)
1942. Perkins, William Harvey, The Lenox, 13th & Spruce Sts. (7)
1946. Perloff, William Harry, Albert Einstein Medical Center (41)
1964. Perrin, George M., First Floor Mills Bldg., Philadelphia General Hospital, 34th & Curie Ave. (19104)
1940. Pessel, Johannes F., 224 West State St., Trenton, N. J.
1952. Peters, Michael, Fairhill Rd., Hatfield, Pa.
1961. Peterson, Lysle H., Bockus Research Institute, 19th & Lombard Sts. (19146)
1940. Pettit, Horace, 601 Suburban Sq. Bldg., Ardmore, Pa.
1949. *Pettit, Mary DeWitt, 3343 W. Penn St. (29)
1946. Pfeiffer, Mildred C. J., 358 Valley Rd., Merion, Pa.
1935. Phillips, Arthur W., 3904 Walnut St. (4)
1961. Phillips, William A., Eastern Pennsylvania Psychiatric Institute (29)
1948. Pierson, Daniel B., Jr., 740 Beacon Lane, Merion, Pa.
1964. Pierucci, Louis, Jr., 1025 Walnut St. (19107)
1961. Pike, Anne H., 5603 Greene St. (44)
1957. *Pilling, George Platt, IV, 2600 N. Lawrence St. (33)

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* S. Weir Mitchell Associate.

1933. *Pillsbury, Donald M., Duhring Labs., University Hospital (19104)
1957. Platt, Ruth M., 441 Lyceum Ave. (28)
1951. Poinsard, Paul J., 2031 Locust St. (3)
1937. Polk, David Stewart, Rosemont, Pa.
1956. *Pontarelli, Domenic J., 1037 Great Springs Rd., Rosemont, Pa.
1953. Porreca, George A., 1912 Spruce St. (3)
1932. Porter, Roland De Lance, Abington Medical Bldg., Abington, Pa.
1951. Post, Joseph W., 105 Glenview Ave., Wyncote, Pa. (19095)
1951. Pote, Harry H., 103 E. Ridley Ave., Ridley Park, Pa.
1958. Potter, H. Phelps, Jr., Woman's Medical College (29)
1951. *Pressman, Robert S., 170 W. Olney Ave. (20)
1965. Price, Joseph J., 36 Copley Road, Upper Darby, Pa.
1947. Prickett, John A., Warrington, Bucks County, Pa.
1954. Prince, Leon N., 2025 Spruce St. (3)
1931. Pryor, Charles Allen, 734 S. Latch's Lane, Marion Station, Pa. (19066)
1952. Pugh, James E., 614 Church Lane, Yeadon, Pa.
1947. Putney, Floyd Johnson, 1712 Locust St. (3)
1943. *Radbill, Samuel X., 7043 Elmwood Ave. (42)
1953. Radbill, Sidney G., Wm. Penn House, 1919 Chestnut St. (19103)
1966. Raffensperger, Bruce W., 8811 Germantown Ave. (19118)
1953. Raffensperger, Edward C., 260 St. James Place (6)
1945. Rakoff, Abraham Edward, Franklin Medical Bldg., 829 Spruce St. (19107)
1953. Ralston, Edgar L., 3400 Spruce St. (4)
1931. Ramsey, Frank M., 620 E. Willow Grove Ave. (18)
1953. Ranieri, Tito A., 2320 S. Broad St. (45)
1941. Rankin, Charles A., Ludlow & Heather Rd., Upper Darby, Pa.
1951. *Rathmell, Thomas K., 446 Bellevue Ave., Trenton, N. J.
1926. *Ravdin, I. S., Univ. of Pa. Hospital (4)
1956. Ravdin, Robert Glenn, 3400 Spruce St. (4)
1959. Raventos, Antolin, 3400 Spruce St. (4)
1962. Raymond, F. Douglas, Jr., Bryn Mawr Medical Bldg., Bryn Mawr, Pa.
1964. Raymond, Samuel, 711 Maloney Bldg., University Hospital, 36th & Spruce Sts. (19104)
1961. Read, William T., Jr., 110 Munn Lane, Cherry Hill, N. J. (08034)
1954. *Reagon, Lindley B., 166 Madison Ave., Mt. Holly, N. J. (08060)
1965. Reddy, John B., 1025 Walnut St. (19107)
1960. Reed, Theodore P., Lankenau Medical Bldg. (19151)
1928. Reese, Warren S., 2118 Locust St. (3)
1919. Reimann, Stanley P., 703 W. Phil-Ellena St. (19)
1964. Reinhard, John J., Jr., 335 Woodstown Road, Salem, N. J.
1956. Reishtein, William A., 16 Blancoyd Rd., Merion Station, Pa.
1963. Reisinger, Paul B., 855 Berkeley Ave., Trenton, N. J.
1951. Rentschler, Laurence B., 5800 Ridge Ave. (28)
1945. *Rhoads, Jonathan E., Univ. of Pa. Hospital (4)
1946. Rhoads, Rebecca M. P., 1014 Thomas Ave., Riverton, N. J. (08077)
1964. Rhoads, S. Creadick, 1014 Thomas Ave., Riverton, N. J. (08077)
1963. Rial, William Y., 111 Dartmouth Ave., Swarthmore, Pa.
1919. Richardson, Russell, Shamona Mill, Downington, Pa.
1942. Riggs, Helena E., 150 Hewitt Rd., Wyncote, Pa.
1936. Ritter, Joseph A., 1034 Bryn Mawr Ave., Narberth, Pa.
1932. Robbins, Frederick R., 317 Milbank Rd., Bryn Mawr, Pa.
1952. Robbins, Robert, 3401 N. Broad St. (40)
1960. Robbins, William S., The Iroquois, Suite 1303, City Line & 47th St. (19131)
1954. *Roberts, Brooke, 3400 Spruce St. (4)
1957. Roberts, Joan Mary, 4 W. Mt. Pleasant Ave. (19)
1964. Robinson, James H., 5900 Spruce St. (19139)
1960. Roeders, Paul H., 330 S. 9th St. (7)
1955. *Rogers, Fred B., 3401 N. Broad St. (40)

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* S. Weir Mitchell Associate.

1951. Ronis, Bernard J., 2106 Spruce St. (3)
1948. Roscoe, Constantine R., 7226 Castor Ave. (24)
1928. Rose, Edward, 426 Owen Rd., Wynnewood, Pa.
1936. Rose, Elizabeth Kirk, 426 Owen Rd., Wynnewood, Pa.
1959. Rose, Isadore, 6000 W. Oxford St. (19151)
1952. Rose, S. Brandt, Chestnut Hill Hospital (18)
1965. Rosenberg, Hyman, 1555 Haddon Ave., Camden, N. J.
1966. Rosenberg, Morton, 1025 Walnut St. (19107)
1946. *Rosemond, George Parrott, 3401 N. Broad St. (40)
1960. Rosenow, Edward C., Jr., American College of Physicians, 4200 Pine St. (4)
1948. Rothman, Maurice M., 1727 Spruce St. (3)
1951. Rouse, George P., Jr., 250 South 18th St. (2)
1963. Rovit, Richard L., Jefferson Medical College (7)
1966. Rovner, Harold, 1427 Spruce St. (19102)
1965. Rowe, Daniel S., Irvine Bldg., Narberth & Montgomery Aves., Narberth, Pa.
1952. Roxby, Bruce S., 5501 Greene St. (44)
1955. Roxby, John B., Jr., 4821 Germantown Ave. (44)
1949. Royster, Henry P., 3400 Spruce St. (4)
1959. Rubin, Alan, 255 S. 17th St. (3)
1952. Rubin, I. Edward, 255 S. 17th St. (3)
1933. Rudolphy, Jay Besson, 312 S. County Line Rd., Wayne, Pa. (19087)
1966. Rugart, Karl F., 811 Spruce St. (19107)
1946. Rupp, Charles, 133 S. 36th St. (4)
1947. Rush, Alexander, 330 S. 9th St. (7)
1963. Rutter, William A., 425 Wister Rd., Wynnewood, Pa.
1963. Ryan, James J., 301 E. City Line, Bala-Cynwyd, Pa.
1945. Rynes, Samuel E., 334 S. 21st St. (3)
1965. Sachs, Marvin L., 133 S. 36th St. (19104)
1952. Sain, Fletcher D., 1245 Highland Ave., Abington, Pa. (19001)
1958. Sall, Manuel, 255 S. 17th St. (3)
1952. Salner, Nathan P., 6812 Castor Ave. (24)
1952. Saltzman, Maurice, 2037 Spruce St. (3)
1966. Samitz, M. H., 1715 Pine St. (19103)
1944. Sampson, David Alan, 726 Braeburn Lane, Penn Valley, Narberth, Pa.
1964. Santangelo, Samuel C., 1941 Woodland Road, Abington, Pa.
1950. Sarner, Joseph B., 289 Locust St. (19106)
1967. Sattilaro, Anthony J., Methodist Hospital (19148)
1954. Savacool, J. Woodrow, 146 W. Tulpehocken St. (44)
1947. Saÿen, John J., 3600 Spruce St. (4)
1948. Scarano, Joseph Albert, 1432 S. Broad St. (46)
1917. *Schaeffer, J. Parsons, 4634 Spruce St. (39)
1929. *Scheffey, Lewis C., 601 Walnut Lane, Haverford, Pa.
1942. *Scheie, Harold G., 3400 Spruce St. (4)
1929. Schenck, Harry P., 1235 Wyngate Rd., Wynnewood, Pa.
1966. Schless, Guy L., 330 South Ninth St. (19107)
1945. Schlezinger, Nathan S., 255 S. 17th St. (3)
1961. Schlosser, Woodrow D., 3701 N. Broad St. (40)
1917. Schnabel, Truman G., 509 Williams Road, Wynnewood, Pa. (19096)
1958. Schnabel, Truman G., Jr., Phila. General Hospital (4)
1957. Schnall, Nathan, 2037 Pine St. (3)
1962. Schneider, Henry C., 4801 Penn St. (24)
1966. Schoenberg, Harry W., 3400 Spruce St. (19104)
1955. Schreader, Charles J., 318 W. Godfrey Ave. (20)
1952. Schulz, Norbert J., 1901 Walnut St. (3)
1963. Schumacher, L. Richard, 60 E. Court St., Doylestown, Pa.
1950. Schumann, Francis, 8811 Germantown Ave. (18)
1948. Schwarz, Gabriel A., 133 S. 36th St. (4)

* S. Weir Mitchell Associate.

1962. Schwarz, Henry P., Philadelphia General Hospital (4)
1955. Schwegman, Cletus W., Hospital of the University of Penna. (4)
1966. Scott, J. Clifford, 826 Maple Ave., Berwyn, Pa.
1930. Scott, John Porter, Children's Hospital, 1740 Bainbridge St. (46)
1950. Scott, Michael, 3401 N. Broad St. (40)
1940. Scott, T. F. McNair, Children's Hospital, 1740 Bainbridge St. (46)
1952. Seifer, Arthur F., 710 South Main St., Phoenixville, Pa.
1958. Sellers, Alfred M., Hosp. of the Univ. of Pa. (4)
1952. Semisch, Charles W., III, 1344 Gilbert Rd., Meadowbrook, Pa.
1967. Seyler, Raymond Q., 1600 Arch St. (19101)
1966. Shaffer, Horace M., 208 W. State St., Trenton, N. J. (08608)
1960. *Shannon, Gerard M., 8118 Bustleton Ave. (15)
1950. Sharp, Reuben L., 719 Cooper St., Camden, N. J.
1963. Sharples, Wynne, 454 S. Ithan Ave., Villanova, Pa. (19085)
1928. Sharpless, Frederick C., 621 Railroad Ave., Haverford, Pa.
1956. Shaw, Daniel L., Jr., 702 Knox Rd., Villanova, Pa.
1963. Shearburn, Edwin W., 306 Lankenau Medical Bldg. (19151)
1955. Schechter, Fred R., 2461 No. 54th St. (31)
1954. Shelly, Walter B., 36th & Spruce Sts. (4)
1955. Shenkin, Henry A., Episcopal Hospital (25)
1946. Shepherd, Samuel Garfield, 1900 Spruce St. (19103)
1956. Sherson, Jacob S., 131 Channing Ave., Malvern, Pa.
1964. Shimkin, Michael B., Temple University School of Medicine (19140)
1936. Shipman, James Shelby, 514 Cooper St., Camden, N. J.
1958. Shipps, Hammell P., 719 Cooper St., Camden, N. J. (2)
1957. Shope, Edward Pierce Lentz, 807 Wilson Bldg., Camden, N. J.
1946. Shoup, George Daniel, 121 W. Walnut Lane (44)
1955. *Shubin, Harry, 6601 N. 21st St. (38)
1952. Shuman, Charles R., 3401 N. Broad St. (40)
1949. Sidlick, David M., Park Towne Place E., 2200 Benj. Franklin Pkwy. (19130)
1941. Silcox, Louis E., Lankenau Medical Bldg. (19151)
1956. *Sillars, Charles H., Abington Memorial Hospital, Abington, Pa.
1963. Silverman, Daniel, 269 South 19th St. (19103)
1955. Silverstein, Alexander, 2114 Pine St. (3)
1966. Simkins, Samuel, 515 W. Godfrey Ave. (19126)
1961. Simmons, Vaughan P., The Fidelity Mutual Life Insurance Co. (1)
1954. Singmaster, Lawrence, 272 Cheswold Lane, Haverford, Pa.
1952. Skromak, Stanley J., 5108 Torresdale Ave. (24)
1929. Smith, Austin T., 330 S. 9th St. (7)
1964. Smith, Edgar C., 7100 Marshall Road, Upper Darby, Pa.
1965. Smith, Eleanor M., 3300 Henry Ave. (19129)
1955. Smith, J. Winslow, 3034 Darby Rd., Ardmore, Pa.
1933. Smith, Lauren Howe, 111 N. 49th St. (39)
1952. Smith, Richard T., 330 S. 49th St. (7)
1967. Smith, Robert R., William H. Rorer, Inc., 500 Virginia Drive, Fort Washington, Pa. (19034)
1964. Smyth, Murray G., Jr., Smith Kline Instrument Co., 1500 Spring Garden St. (19101)
1951. Snape, William J., 573 Stevens St., Camden, N. J.
1966. Snow, Laurence H., 3300 Henry Ave. (19129)
1953. Snyder, G. Gordon, Bryn Mawr Medical Bldg., Bryn Mawr, Pa.
1958. Sodeman, William A., 1025 Walnut St. (7)
1961. Soffe, Alvin M., 1930 Chestnut St. (3)
1935. Sokoloff, Martin J., 255 S. 17th St. (3)
1964. Soll, David B., 5001 Frankford Ave. (19124)
1940. Soloff, Louis A., 3401 N. Broad St. (40)
1950. Somerville, William J., 37 S. 20th St. (3)
1944. Sommer, George N. J., Jr., 120 W. State St., Trenton, N. J.

* S. Weir Mitchell Associate.

1952. Sones, Maurice, Mt. Airy Medical Bldg. (19)
1951. Sonneborn, Duane G., 1200 W. Erie Ave. (24)
1928. Spaeth, Edmund B., 1930 Chestnut St. (3)
1966. Spaeth, George L., 1930 Chestnut St. (19103)
1958. Spaeth, Philip G., 1930 Chestnut St. (3)
1937. Spangler, John Luther, Devon, Pa.
1957. Spelman, Joseph Worcester, Dept. of Public Health, 13th & Wood Sts. (7)
1935. Spiegel, Ernest A., 6807 Lawnton Ave. (26)
1961. *Spiegelman, Jay, Benson East Apt., Jenkintown, Pa.
1955. Spitz, Eugene B., 40–50 W. Front St., Media, Pa.
1958. Splendido, Joseph A., 608 W. Cliveden (19)
1961. Stahlgren, LeRoy H., Episcopal Hospital, Front & Lehigh Ave., (19125)
1960. Stainback, William C., Bryn Mawr Medical Bldg., Bryn Mawr, Pa.
1957. Stanley, Robert C. G., 2315 Edgemont Rd., Chester, Pa.
1927. Starr, Isaac, 505 Cresheim Valley Rd. (18)
1952. Stauffer, Herbert M., 3401 N. Broad St. (40)
1952. Stayman, Joseph W., Jr., 8815 Germantown Ave. (18)
1945. Stecher, H. Armin, 1510 Darby Rd., Havertown, Pa.
1955. Steel, Howard H., 238 W. Allens Lane (19119)
1952. *Steiger, William A., 3401 N. Broad St. (40)
1964. Stein, Donald B., Jr., Fitzgerald Mercy Hospital, Darby, Pa.
1946. Stein, Irvin, 1936 Spruce St. (3)
1966. Stein, Joseph M., 414 Cooper St., Camden, N. J. (08102)
1953. Stein, Raymond O., 269 S. 19th St. (3)
1966. Stein, Samuel C., 1930 Chestnut St. (19103)
1963. Steinberg, Stanley B., 2461 N. 54th St. (19131)
1936. Steinfield, Edward, 2200 Benjamin Franklin Parkway (30)
1951. Sterling, Julian A., 1008 Sharpless Rd. (26)
1948. Stevens, Lloyd W., 133 S. 36th St. (4)
1928. *Stokes, Joseph, Jr., Henry Phipps Institute, 4219 Chester Ave. (19104)
1943. Stokes, S. Emlen, 129 Chester Ave., Moorestown, N. J.
1961. Strang, John E., 330 S. 9th St. (7)
1953. Strawbridge, Rendall R., Lankenau Medical Bldg. (19151)
1937. *Strumia, Max M., Penn Valley, Narberth, Pa.
1960. *Strumia, Paul V., Bryn Mawr Hospital, Bryn Mawr, Pa.
1951. *Sturgis, Katharine Boucot, Woman's Medical College, Henry Ave. & Abbottsford Road (19129)
1927. Sturgis, Samuel Booth, 349 Wistar Rd., Wynnewood, Pa.
1930. Sunderman, F. William, 1833 Delancey Place (3)
1966. Sullivan, Howard E., Jr., Bryn Mawr Medical Bldg., Bryn Mawr, Pa. (19010)
1954. *Sussman, Marcel S., Wm. Penn House, 1919 Chestnut St. (19103)
1963. Sutliff, Frederick P., 3701 N. Broad St. (19140)
1965. Sutnick, Alton I., 2135 St. James Place (19103)
1965. Swartley, Robert N., 8815 Germantown Ave. (19118)
1952. Talbot, Timothy R., Jr., 204 Booth Lane, Haverford, Pa.
1963. Tassman, William S., 626 Four Penn Center Plaza, 16th & J. F. Kennedy Blvd. (19103)
1946. Tauber, Robert, 2019 Walnut St. (3)
1952. Taylor, Ann Gray, 6364 Germantown Ave. (44)
1960. Taylor, Daniel B., 2241 Federal St. (46)
1965. Taylor, Richard C., 11th & Sansom Sts. (19107)
1956. Templeton, John Y., III, 805 Spruce St. (19107)
1966. Teplick, Joseph G., 1351 W. Tabor Road (19141)
1966. Terry, Luther L., 121 College Hall, Univ. of Pennsylvania (19104)
1951. Theodos, Peter A., 1930 Chestnut St. (3)
1961. Therman, Per-Olof, 111 N. 49th St. (39)
1959. Thomas, Arthur H., 5240 Vine St. (39)
1946. Thomas, Carmen C., 1930 Chestnut St. (3)

* S. Weir Mitchell Associate.

1964. Thomas, John W., 5900 Spruce St. (19139)
1952. Thompson, Charles M., 255 S. 17th St. (3)
1927. Thorington, J. Monroe, Cambridge Apts., Wissahickon Ave. & School House Lane (44)
1936. Thudium, William J., 700 Merion Square Rd., Gladwyne, Pa.
1954. Tindall, Dorohy D., 1930 Rittenhouse Sq. (3)
1966. Toland, Joseph J., III, 3273 Teesdale St. (19136)
1935. Toland, Owen J., 336 Aubrey Rd., Wynnewood, Pa.
1952. *Tondreau, Roderick L., 523 Old Gulph Rd., Bryn Mawr, Pa.
1949. Tornay, Anthony S., 2038 Locust St. (3)
1966. Torrance, Edward G., 678 Burmont Rd., Drexel Hill, Pa.
1936. Towson, Charles Emory, 224 W. Washington Lane (44)
1955. Trimpi, Howard D., 501 N. 17th St., Allentown, Pa.
1954. Trommer, Philip R., 258 S. 18th St. (3)
1956. Troncelliti, Mario V., Pennsylvania Hospital (7)
1953. Tropea, Frank, Jr., 500 Mulberry Lane, Haverford, Pa.
1947. Trueman, Robert H., 2101 Chestnut St. (3)
1954. Truitt, R. Marshall, Jr., 6400 Wissahickon Ave. (19119)
1965. Tsaltas, Theodore T., 1025 Walnut St. (19107)
1966. Tucker, Gabriel F., Jr., 3401 N. Broad St. (19140)
1959. Tuddenham, William J., Pennsylvania Hospital (7)
1938. Tuft, Louis, 1530 Locust St. (2)
1964. Tulsky, Emanuel G., Abington Memorial Hospital, Abington, Pa. (19001)
1933. *Tumen, Henry J., 1830 Rittenhouse Square (3)
1938. Turman, Christopher M., 1245 Highland Ave., Abington, Pa. (19001)
1923. Turner, Creighton H., 1731 Pine St. (3)
1954. Turner, Linton W., 3401 N. Broad St. (40)
1959. Tyson, R. Robert, 3401 N. Broad St. (40)
1937. *Uhle, Charles A. W., Lankenau Medical Bldg. (19151)
1953. Ulin, Alexander W., 230 N. Broad St. (2)
1960. Uricchio, Joseph F., 245 N. Broad St. (7)
1950. Valdes-Dapena, Antonio, 214 Plush Mill Rd., Wallingford, Pa.
1958. Van den Noort, Gordon S., 888 Glenbrook Ave., Bryn Mawr, Pa.
1937. *Vander Veer, Joseph B., 330 S. 9th St. (7)
1937. Van Loon, Emily Lois, 255 S. 17th St. (3)
1960. Van Meter, Ralph H., 244 W. Main St., Moorestown, N. J.
1933.**Vastine, Jacob H., 2nd, 267 Kent Rd., Wynnewood, Pa.
1967. Vaughan, Victor C., III, 2600 N. Lawrence St. (19133)
1963. Vaughan, Arthur R., Jr., 5329 Rising Sun Ave. (19120)
1958. Velkoff, Cyril L., 2100 Walnut St. (3)
1965. Viggiano, Louis X., 301 Long Lane, Upper Darby, Pa.
1952. Vischer, Thomas J., 5903 Greene St. (44)
1940. Voegelin, Adrian W., Germantown Professional Bldg. (44)
1960. Wagenheim, Harry H., 1822 Spruce St. (3)
1949. Wagner, Frederick B., Jr., 800 Chauncey Road, Narberth, Pa.
1949. Wagner, Joseph A., 201 Highland Ave., Bryn Mawr, Pa.
1938. Waldman, Joseph, 404 Meadowbrook Lane (18)
1955. Waldron, Jerome M., Evergreen Towers, 8900 Roosevelt Blvd. (19115)
1956. Watkins, E. Lloyd, 8811 Germantown Ave. (18)
1952. Weaver, Harry S., Jr., 37 S. 20th St. (3)
1953. Weber, Lennard L., 829 Spruce St. (19107)
1952. Weinstein, George L., 255 S. 17th St. (3)
1950. Weiss, Sidney, 2037 Locust St. (3)
1965. Weiss, William, Philadelphia General Hospital, 34th & Curie Ave. (19104)
1959. Wellenbach, Burton L., 1930 Chestnut St. (19103)

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* S. Weir Mitchell Associate.
** Military Service

1951. *Welty, Jack W., Montgomery Ave. & Owen Rd., Wynnewood, Pa.
1955. Wendkos, Martin H., Green Hill in Lower Merion (19151)
1962. Wentz, Clarkson, 20 S. Valley Road, Paoli, Pa.
1961. Wermuth, William C., 414 Minden Way, Wynnewood, Pa. (19096)
1959. West, Clifton F., Jr., 406 Lankenau Medical Bldg. (19151)
1921. Whitaker, William, 4645 Oakland St. (24)
1936. White, Ellen Pawling Corson-, Gladwyne, Pa.
1965. White, Joseph C., Barrow Neurological Institute of St. Joseph's Hospital, 302 Thomas Rd., Phoenix, Arizona (85013)
1960. Whiteley, William H., 1015 Chestnut St. (7)
1925. Widmann, Bernard P., 250 S. 18th St. (3)
1954. Wieder, Henry S., Jr., 330 S. 9th St. (7)
1939. *Willauer, George, 6129 Greene St. (44)
1937. *Williams, John C., 6370 Germantown Ave. (44)
1965. Williams, Kirkley, R., Bryn Mawr Medical Bldg., Bryn Mawr, Pa. (19010)
1920. Williams, Philip F., Broughton Lane, Villanova, Pa.
1950. Williams, R. G., Univ. of Pa. School of Medicine (4)
1927. Williamson, Ernest G., 6353 Woodbine Ave. (19151)
1945. Wilson, John F., Fairmount Farm, 561 Fairthorne Ave. (19128)
1949. Wilson, William W., 133 S. 36th St. (4)
1957. Winkleman, N. William, Jr., 4141 Apalogen Rd. (44)
1963. Winston, Joseph M., Jeanes Hospital (11)
1941. Wirts, C. Wilmer, 2017 Delancey St. (3)
1966. Wise, John S., 433 Bellevue Ave., Trenton, N. J. (08618)
1961. Wise, Robert I., 1025 Walnut St. (7)
1953. Wohl, George T., Philadelphia General Hospital (4)
1933. Wohl, Michael G., 1727 Pine St. (3)
1958. Woldow, Irving, Einstein Med. Center, York & Tabor Rds. (41)
1958. Wolf, John H., 124 W. Walnut Lane (44)
1944. Wolf, Lewis R., 3474 Frankford Ave. (34)
1962. Wolferth, Charles C., Jr., The Windsor, Suite 2307, 1700 Benj. Franklin Pkwy. (19103)
1964. Wolfson, Robert J., 3701 N. Broad St. (19140)
1964. *Wolgin, William, 1512 Spruce St. (19102)
1957. Wolkowicz, Michael I., 2022 Spruce St. (3)
1942. Wolman, Irving J., 7607 Woodlawn Ave. (26)
1966. Wood, Alfred C., Jr., 6386 Church Rd. (19151)
1932. *Wood, Francis C., 3400 Spruce St. (19104)
1944. Wood, H. Curtis, Jr., 7010 Lafayette Ave., Fort Washington, Pa.
1966. Wood, Margaret G., 3400 Spruce St. (19104)
1953. *Woodruff, Marston T., 4940 Penn St. (24)
1928. Wright, Carroll S., 1402 Spruce St. (2)
1960. Wurzel, Harold A., 3400 Spruce St. (4)
1966. Yaskin, H. Edward, 589 Stevens St., Camden, N. J. (08103)
1964. Young, Alma L., 3343 W. Penn St. (19129)
1941. *Young, Barton R., Germantown Hospital (44)
1966. Young, Irving, Albert Einstein Medical Center, N.D. (19141)
1961. Zanni, Anthony L., 111 N. 49th St. (39)
1957. Zaslow, Jerry, 1351 Tabor Rd. (41)
1955. Zatuchini, Jacob, 3401 N. Broad St. (40)
1946. Zeigerman, Joseph H., 2105 Spruce St. (3)
1949. Zintl, William J., 69th & Market Sts., Upper Darby, Pa.
1956. Ziserman, Abraham J., 2046 Pine St. (3)
1962. Zubrow, Sidney N., 521 Pine St. (6)
1933. Zulick, J. Donald, 307 Fisher Rd., Jenkintown, Pa. (19046)
1965. Zweiman, Burton, Univ. of Pa. Hospital (19104)

---

* S. Weir Mitchell Associate.

Non-Resident Fellows

ELECTED

1936. Alexander, Fay K., Lincolnville, Maine
1953. Allman, David B., 4105 Brigantine Blvd., Brigantine, N. J. (08203)
1941. Aronson, Ronald S., 9401 Byeforde Road, Kensington, Md. (20795)
1954. Austin, George M., University of Oregon Medical School, Portland, Oregon
1942. Bachman, Carl, 1337 Andrea Ave., St. Helena, Cal. (94574)
1958. Ballinger, Walter F., II, Johns Hopkins Hospital, 1900 E. Monument Ave., Baltimore, Md. (21205)
1955. Beckfield, William J., Luther Hospital, 310 Chestnut St., Eau Claire, Wisconsin
1949. *Beecham, Clayton T., Geisinger Medical Center, Danville, Pa.
1932. Behney, Charles A., P. O. Box 4256, San Jose Branch, Bisbee, Arizona.
1955. Blank, Harvey, Jackson Memorial Hospital, Miami 36, Fla.
1921. Borzell, Francis F., R. D. 2, Box 2051, Browns Mills, N. J.
1937. Braceland, Francis J., The Institute of Living, 200 Retreat Ave., Hartford, Conn.
1951. Brinton, S. Jervis, 3 Gulf View Rd., Palm Beach, Fla. (33482)
1948. *Brown, vadm. Robert B., mc, usn, U. S. Naval Hospital, Bethesda, Md.
1959. Brownelller, Ellsworth R., Geisinger Medical Center, Danville, Pa.
1953. Burbridge, J. Raymond, 163 Nassau St., Princeton, N. J.
1952. Bumgardner, Heath D., 13 S. Main St., Cape May Court House, N. J.
1931. Cantarow, Abraham Dept. of Health, Education & Welfare, NIH, Bethesda, Md. (20014)
1948. Chapman, John P., Calle Fray Junipero Serra 26, Palma de Mallorca, Balearic Islands, Spain
1950. Chapple, Charles C., 8517 Indian Hills Drive, Omaha, Neb. (68114)
1941. Cheleden, John, 1012 Volusia Ave., Daytona Beach, Fla.
1930. Clarke, Francis Mann, 116 New St., New Brunswick, N. J.
1926. Clerf, Louis H., 5575-8th Ave., North St. Petersburg, Fla.
1964. Cohen, Sheldon G., 309 S. Franklin St., Wilkes-Barre, Pa.
1930. Cottrell, James E., 27 Shinnecock Trail, Medford Lakes, N. J.
1959.**Cowan, RADM. John S., MC, USN, Pacific Fleet Surgeon, Hdq., U.S. Pacific Fleet, FPO, San Francisco, Calif. (96601)
1960. Cowley, Allen W., Sr., 1919 N. Front St., Harrisburg, Pa.
1947. Cutler, Norman L., 1300 Harrison St., Wilmington, Del.
1941.**Cuttle, Tracy D., U. S. Naval Hospital, Chelsea, Mass. (02150)
1959. Dannenberg, Arthur M., Jr., Johns Hopkins School of Hygiene, 615 N. Wolfe St., Baltimore, Md. (21205)
1955. Daugherty, J. Arthur, 1511 N. Front St., Harrisburg, Pa.
1937. Davis, Perk Lee, R. D. 5, Waynesburg, Pa. (15370)
1954. Deitrick, John E., Cornell Univ. Medical College, 1300 York Ave., New York 21, N. Y.
1941. Dixon, Harold M., 4805 46th St., N.W., Washington 16, D. C.
1953. Dixon, James P., Antioch College, Yellow Springs, Ohio
1961. Duane, Thomas D., 35 E. Elizabeth Ave., Bethlehem, Pa.
1959. Durgin, Bernice Elise, 221 N. Allegheny St., Bellefont, Pa.
1947. Durkin, John K., V.A. Hospital, Coatesville, Pa. (19320)
1964. Eckenhoff, James E., Northwestern Univ. Medical School, Chicago, Ill. (60611)
1955. Edwards, Margaret Hay, 800 4th St., S.W., Washington 24, D. C.
1953. Eisenberg, Isadore J., 2806 Green St., Harrisburg, Pa.
1951. Ellis, Mackinnon, V.A. Hospital Martinez, Cal.
1957.**Errion, Arthur Robbins, U. S. Naval Hospital, Fleet Post Office, San Francisco, Cal. (96662)
1951. Ervin, Carl E., 240 North Third Street, Harrisburg, Pa.
1944. Estes, W. L., Jr., 35 E. Elizabeth Ave., Bethlehem, Pa.
1954. Evans, Harry D., Jr., American Cyanamid Co., 1 Caspar St., Danbury, Conn.

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* S. Weir Mitchell Associate.

1952. Faller, Constantine P., 127 State St., Harrisburg, Pa.
1952. Flinn, Lewis B., 1306 Broom St., Wilmington 6, Del.
1940. Forrester, James S., Harrisburg Polyclinic Hospital, Harrisburg, Pa.
1951. Friedland, Carl K., 271 W. Blackwell St., Dover, N. J.
1947. Gaskill, Herbert S., 9648 E. Powers Drive, Englewood, Colo.
1913. Gates, Nathaniel, 2240 W. Grand Blvd., Detroit 8, Mich.
1958. Geckeler, George D., Tranquility, S. Windham, Vt.
1957. Giering, John Fleming, 84 James St., Kingston, Pa.
1931. Gilman, Robert Louis, Cheyney, Pa.
1954. Godfrey, Ellwood W., Princeton Hospital, Princeton, N. J.
1928. Gordon, Burgess L., 1550 Lake Shore Dr., Chicago, Illinois
1952. Grieco, Reynold M., 2 W. 4th St., Williamsport, Pa.
1934. Griffith, John Q., Jr., 6 North Fredericksburg Ave., Margate, N. J.
1952. Gross, Elmer R., Medical Arts Bldg., Wilmington, Del.
1933. Gruber, Charles M., 1313 College Ave., Redlands, Calif.
1951. Hafkenschiel, Joseph H., Jr., 450 Sutter St., San Francisco, Cal. (94108)
1935. Haines, Harlan F., 231 N. Pine St., Seaford, Del.
1939. Hamblock, Leonard C., Masonic Homes, Elizabethtown, Pa. (17022)
1959. Hampton, Louis J., 300 Highland Ave., Hanover, Pa. (17331)
1955. Harris, John H., 1301-A N. Second St., Harrisburg, Pa.
1965. Harris, John H., Jr., Carlisle Hospital, Carlisle, Pa. (17013)
1932. Harris, Stanley E., 229 Maple Ave., Delanco, N. J.
1936. Hatch, Lerleen Clement, 1642 Saddle Hill Drive, Logan, Utah
1947. *Hawthorne, Herbert R., 1016 Yardley Rd., Morrisville, Pa.
1928. Hayman, Joseph M., Jr., Tufts University Medical School, Boston, Mass.
1946. Hendrickson, Frank O., Jupiter, Fla.
1955. Hills, Arthur G., 1000 N. W. 17th St., Miami, Fla. (36)
1933. Hitzrot, Lewis H., 161 Patton Ave., Princeton, N. J. (08540)
1950. Horn, Robert C., Henry Ford Hospital, Detroit, Mich.
1953. Imber, Irving, 428 Walnut St., Reading, Pa.
1958. Impink, Robert R., 405 Oley St., Reading, Pa.
1935. Ingleby, Helen, The Church House, Heacham, King's Lynn, England
1921. *Ivy, Robert H., 104 Dalton Rd., Paoli, Pa.
1955. *Jordan, Charlotte B., 175 E. Brown St., E. Stroudsburg, Pa.
1955. Jordan, Claus G., 175 E. Brown St., E. Stroudsburg, Pa.
1936. Kain, Thomas M., 30 Northeast 97th St., Miami, Fla.
1956. Keffer, William H., 413 Oley St., Reading, Pa.
1947. Kelchner, Clyde H., 1125 Turner St., Allentown, Pa.
1954. LaMotte, William O., Jr., Professional Bldg., Wilmington 3, Del.
1936. Leaman, William G., Jr., P. O. Box 463, Unionville, Pa. (19375)
1943. Leopold, Irving H., The Mount Sinai Hospital, 100th St. & 5th Ave., New York, N, Y. (10029)
1952. Levan, John B., 401 Oley St., Reading, Pa.
1960. Lewis, David H., Hogenskildsgatan 13, Göteborg Ö, Sweden
1949. Lhamon, William T., The New York Hospital, 525 E. 68th St., New York, N. Y. (10021)
1954. Light, John J. B., 25 N. 9th St., Lebanon, Pa.
1954. Lindenmuth, Woodrow W., V.A. Hospital, West Haven, Conn.
1948. Livingood, Clarence S., Henry Ford Hospital, Detroit 2, Mich.
1932. *Lukens, Francis D. W., Veterans Administration Hospital, University Drive, Pittsburgh, Pa. (15240)
1962. McGregor, Robert A., The Professional Bldg., Augustine Cut-Off, Wilmington 3, Del.
1948. McLaughlin, John T., 655 N. Central Ave., Glendale, Calif.
1950. Mackmull, Gulden, 174 Nob Hill Lane, Ventura, California
1966. Maguire, Henry C., Jr., Rockefeller University, 66th St. & York Ave., New York, N.Y. (10021)
1942. Manges, Lewis C., Jr., Reed Memorial Hospital, Hancock, N. Y.

* S. Weir Mitchell Associate.

1959. Maniglia, Rosario, Holy Spirit Hospital, Camp Hill, Pa.
1953. Margolies, Michael, 567 E. Chestnut St., Coatesville, Pa.
1955. Margolies, M. Price, 379 E. Chestnut St., Coatesville, Pa.
1935. Mason, James B., 1730 S. Jackson St., Tacoma, Washington
1932. Meade, Richard H., Jr., 750 San Jose Dr., Grand Rapids, Mich.
1936. Middleton, William Shainline, 2114 Adams St., Madison, Wisc.
1946. Miller, Franklin R., Snyder-Jones Clinic, First Nat. Bk. Bldg., Winfield, Kansas
1949. Motley, Hurley L., 1212 Shatto St., Los Angeles, Calif.
1949. *Mulholland, Stanford W., P.O. Box C, Christiansted, St. Croix, Virgin Islands (00820)
1960. Ojers, Gaylord W., 35 E. Elizabeth Ave., Bethlehem, Pa.
1956. Oppenheimer, Albert, 224 E. Main St., Moorestown, N. J.
1926. Paul, John Rodman, 333 Cedar St., New Haven, Conn.
1959. Pendergrass, Henry P., Massachusetts General Hospital, Boston, Mass.
1950. Pincus, Irwin J., 610 N. Roxbury Drive, Beverly Hills, Calif.
1958. Rachlin, Lillian, 370 Golden Oak Drive, Portola Valley, Calif. (94026)
1966. Rahn, Elsa Kertesz, 363 Hempstead Ave., Rockville Center, N.Y. (11570)
1938. Read, Hilton S., 5407 Atlantic Ave., Atlantic City, N. J.
1964. Reed, Miriam, 253 Witherspoon St., Princeton, N. J.
1958. Reinhardt, David J., III, 1303 Delaware Ave., Wilmington, Del.
1936. Reisinger, John A., 1835 Eye St., N.W., Washington, D. C.
1955. Rettew, Philip L., 612 Museum Rd., Reading, Pa.
1951. Richardson, Fred MacD., Bureau of Medical Review, 84 Holland Ave., Albany, N. Y. (12208)
1961. Richardson, Herschel E., 205 Mayerline St., Houston, Texas
1947. Rogers, Arthur Merriam, 1351 Durwood Drive, Charlotte, N. C.
1941. Royster, Hubert A., Jr., Watts Hospital, Durham, N. C. (27705)
1954. Sampson, Martin C., Wallace Laboratories, Cranbury, N. J.
1936. Sands, Joseph Evans, 135 107th St., Stone Harbor, N. J.
1945. Schell, James F., 506 Sharpley Lane, Wilmington, Del.
1958. Schreck, Kenneth M., 1527 Meadowlark Road, Wyomissing, Pa. (19610)
1921. Schumann, Edward A., 4141 Jackson Drive, Lafayette Hill, Pa.
1951. Seitchik, Joseph, Sinai Hospital of Baltimore, Baltimore, Md.
1941. Shands, Alfred R., Jr., Alfred I. duPont Institute, P.O. Box 269, Wilmington, Del. (19899)
1964. Sharpe, William D., New Jersey College of Medicine, Jersey City 4, N. J.
1950. Shiffer, Paul H., Dreher Ave., R.D. #2, Stroudsburg, Pa.
1951. Shorey, Winston K., 7117 Shamrock Drive, Little Rock, Arkansas
1933. Smelzer, Donald C., 207 E. Orange St., Lancaster, Pa.
1954. Smith, Charles W., 128 State St., Harrisburg, Pa.
1927. Smyth, Calvin M., Hartsville, Bucks Co., Pa. (18974)
1929. Sponsler, Marshall B., 103 Lansdowne Road, DeWitt, N. Y.
1942. Stengel, Alfred, Jr., Maple Ave., Katonah, N. Y.
1955. Stevenson, Theodore D., 475 Riverside Drive, New York, N. Y. (10027)
1934. Stewart, Sloan G., 6200 Atlantic Ave., Ventnor, N. J.
1931. Stiles, Merritt H., E. 406 14th Ave., Spokane 3, Wash.
1953. Stroud, M. W., Shaker Blvd., Shagrin Falls, Ohio (44022)
1934. Summey, Thomas J., Morrowdale Farm, Rt. 2, Charlottesville, Va.
1961. Sunderman, F. William, Jr., University of Florida College of Medicine, Gainesville, Fla. (32603)
1952. Sweeney, Frances X., Station A, Marlboro, N. J. (07746)
1931. Taylor, Norman H., Queen of the East, E. Broadway, Hamilton, Bermuda
1928. Thomas, J. Earl, Loma Linda University, Loma Linda, Calif.
1942. Thompson, Wesley D., 247 E. 3rd St., Lewistown, Pa.
1941. Tompkins, Pendleton, 490 Post St., San Francisco, Calif.
1897. Veasey, Clarence A., 1118 W. 9th Ave., Spokane 9, Wash.
1941. Weaver, Ruth H., 108 Atlantic Ave., Atlantic City, N. J.

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* S. Weir Mitchell Associate.

1957. Webster, Marie, 1623 Hyde St., San Francisco 9, California
1966. Wentzler, J. Donald, Divine Providence Hospital, Williamsport, Pa. (17701)
1959. Whayne, Tom F., University of Kentucky Medical Center, Lexington, Ky.
1966. White, Richard K., 1702 Walnut St., Allentown, Pa. (18104)
1936. Wilder, Theodore S., Linden Lane, Sherborn, Mass.
1935. Willard, John H., Harlan Memorial Hospital, Harlan, Kentucky
1949. Willson, J. Robert, University of Michigan Medical Center, Ann Arbor, Michigan (48104)
1948. Winston, Julius, 1616 Pacific Ave., Atlantic City, N. J. (08400)
1963. Wood, Horatio C., IV, Cincinnati General Hospital, Cincinnati, Ohio
1953. Zarafonetis, Chris J. D., Simpson Memorial Institute, University of Michigan, Ann Arbor, Michigan

Honorary Fellows

Limited to fifty, of whom twenty may be foreigners.

American

ELECTED

1964. Corner, George Washington, M.D., SC.D.(HON.), LL.D.(HON.), LITT.D.(HON.), 1810 Rittenhouse Square (3)
1959. Fay, Marion S., M.A., PH.D., SC.D.(HON.), LL.D.(HON.), Women's Medical College, Henry Ave. & Abbottsford Rd. (29)
1965. Gurin, Samuel, PH.D., SC.D.(HON.), University of Pennsylvania School of Medicine, 36th and Hamilton Walk (19104)
1958. Shryock, Richard H., PH.D., LL.D.(HON.), American Philosophical Society, Independence Square (6)

Foreign

1937. Dale, Sir Henry Hallet, C.B.E., F.R.S., O.M., M.D., F.R.C.P., 52 Queen Anne St., London, W. 1, England
1955. Mann, Ida, M.A., D.SC., M.B., B.S., L.R.C.P., D.O.M.S., F.R.C.S., Ophthalmic Consultant to the Government of W Australia, 56 Hobbs St, Nedlands, Perth, W. Australia.

Honorary Associate Fellows

1955. Diemand, John A., President, Insurance Company of North America, 1600 Arch St. (2)
1964. Pew, J. Howard, Grays Lane & Mill Creek Rd., Ardmore, Pa.

Necrological List

Resident Fellows	*Date of Election*	*Date of Death*
1. Brooke, John A.	January 7, 1953	January 28, 1967
2. Colonna, Paul C.	January 13, 1943	June 7, 1966
3. Crossan, Edward T.	May 4, 1921	September 11, 1966
4. Deibert, Irvin E.	January 7, 1935	January 5, 1967
5. *Edeiken, Joseph	January 11, 1937	December 30, 1966
6. Fetter, Theodore R.	January 7, 1935	January 19, 1967
7. Frankel, Kalman	April 1, 1959	May 6, 1966
8. *Gold, David A.	December 5, 1962	April 21, 1966
9. Houser, Karl M.	January 9, 1928	January 5, 1967
10. Krauss, Frederick Harold	January 11, 1944	July 17, 1966

* S. Weir Mitchell Associate.

11.	Krumbhaar, Edward B.	May 6, 1914	March 16, 1966
12.	Lurie, Max B.	January 8, 1947	September 23, 1966
13.	Macfarlan, Douglas	January 13, 1943	September 12, 1966
14.	Novak, Paul	October 2, 1963	February 8, 1966
15.	Outerbridge, George W.	October 1, 1913	January 16, 1967
16.	Piersol, George Morris	June 7, 1911	August 19, 1966
17.	Richardson, Russell	October 1, 1919	July 2, 1966
18.	Robertson, Harold F.	May 9, 1932	December 30, 1966
19.	Roche, Philip Q.	October 12, 1938	July 30, 1966
20.	Sautter, Albert C.	March 4, 1908	November 13, 1966
21.	Sheridan, Joyce T.	January 11, 1932	August 4, 1966
22.	Tassman, Isaac S.	January 13, 1943	June 10, 1966
23.	Walking, Aldolph A.	May 9, 1932	March 28, 1966
24.	Weaver, Robert L.	January 5, 1955	May 15, 1966
25.	Weeder, S. Dana	May 12, 1930	October 5, 1966

Non-Resident Fellows		*Date of Election*	*Date of Death*
1.	Hershey, J. Ivan	October 4, 1944	February 13, 1966
2.	La Motte, William O.	October 4, 1944	October 5, 1966
3.	Nicholson, Samuel T., II.	January 11, 1937	May 8, 1966

Honorary Associates		*Date of Election*	*Date of Death*
1.	Clement, Martin W.	March 2, 1955	August 30, 1966

Honorary-American		*Date of Election*	*Date of Death*
1.	Richards, Alfred Newton	January 6, 1936	March 24, 1966
2.	Stone, Robert Spencer	January 8, 1947	December 18, 1966

(Suggested Form of Bequest)

I hereby give, devise, and bequeath to The College of Physicians of Philadelphia, a charitable and educational association organized under the laws of the Commonwealth of Pennsylvania and the executive offices whereof are located in the City of Philadelphia, the sum of.....

a. which sum shall be used for the general purposes of said association;

b. which sum shall be added to and become a part of the general endowment fund of said association;

c. which sum shall be used by said association preferably for the following purposes:

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